COLORECTAL MALIGNANCIES
A COMPREHENSIVE STUDY

Dissertation Submitted

for the Degree of

MASTER OF SURGERY

Branch I

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COIMBATORE MEDICAL COLLEGE

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CERTIFICATE

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Degree of MASTER OF SURGERY

Branch I (GENERAL SURGERY)

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DECLARATION

I solemnly declare that this Dissertation on **"COLORECTAL MALIGNANCIES A COMPREHENSIVE STUDY"** was done by me at Coimbatore Medical College Hospital, Coimbatore under the guidance and supervision of **DR.A.RAMA MOORTHY, M.S.**

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INTRODUCTION AND HISTORICAL DATA

Colorectal cancers though more common in the west are on the increase in our country for the past decade. The early detection of this disease is of paramount importance in its outcome. Few topics in cancer research have engendered more excitement than the recent discovery of identifiable genetic defect in patients with inherited as well as sporadic form of colorectal carcinoma.

There are evidences that neoplastic disease has affected humans since prehistoric times. Mummies from Pre-Columbian, Peru of 2400 years ago as well as Egyptian mummies from 3000 B.C. have metastatic skeletal deposits. It was Hippocratic (460 – 370 B.C.) who first propose a theoretical framework to explain cancer invasion.

The Cellular etiology of cancer was first described by Johannes Peter Mueller in 1828. The following year Joseph calrude Reaemer proposed that invasion and distant spread were the result of translocation of cells and he coined the term metastasis. The first successful resection of colonic growth was performed by Reynoard lyons in 1823.

After Billroth, Czerny and Mikulicz, the pioneers in abdominal surgery familiarized, the technique of intestinal resection and anastamosis, increasing number of colonic resection were attempted. The combined operation involving abdominal and perineal phases for excision of the rectum was first performed by Czerny (1883). But it was undoubtedly the work of Ernest Miles (1908) who established the abdomino perineal operation.

Cuthberk Dukes (1935) classified carcinomas of rectum into 3 stages and explaining macroscopic variations. These are widely used by pathologists with minimal changes even now for colorectal cancer staging Paul of Liverpool (1895) and Mikulicz of Brestan (1903) devised extra peritoneal resection of carcinoma colon and popularized the technique in America.

Halstead (1895), Shoemaker (1921), Rankin (1928) and Wangensteen (1940) developed various method of anastamosis by which it was hoped to carry out resection and anastamosis in an entirely sterile manner without opening the bowel lumen till union was completed. But is was later pointed out by Moynihan that the factor responsible for sepsis is not contaminated during the operation itself but subsequent leakage.

Whipple (1931) and Turner (1937) favoured intraperitoneal resection with temporary caecostomy in order to relieve the tension on the suture lines. Devine (1931) developed preliminary defunctioning colostomy which helped mechanical cleansing of the distal bowel.

After advent of strong intestinal antiseptics reliance was paced on them entirely and a primary colostomy was entirely omitted. Lloyd Davis Morgan and Yollinger (1953) carried out resection with immediate anastamosis without any form of proximal decompression. In their series of 109 cases, there were only 3 postoperative death and none of them due to sepsis.

In recent years, the trend is towards preparation with mechanical cleansing using balanced salt solutions containing osmotic purgatives in them with antibiotics, orally or IV. This requires only single day preoperative preparation.

Surgical resection remains the mainstay of treatment for colorectal cancer. Radiotherapy and chemotherapy are used as adjuvant therapeutic options. Turnball at the Cleveland clinic recommended a no touch technique in which vascular and mesenteric division was first undertaken, thereby isolating the tumour.

The role of gene and their abnormality are being studied extensively and had given us the adenoma to carcinoma model due to

accumulation of various genetic defects in the form of deletion, translocation etc. These may help us to find appropriate diagnostic tool to look for such aberration and early prevention of cancer progression.

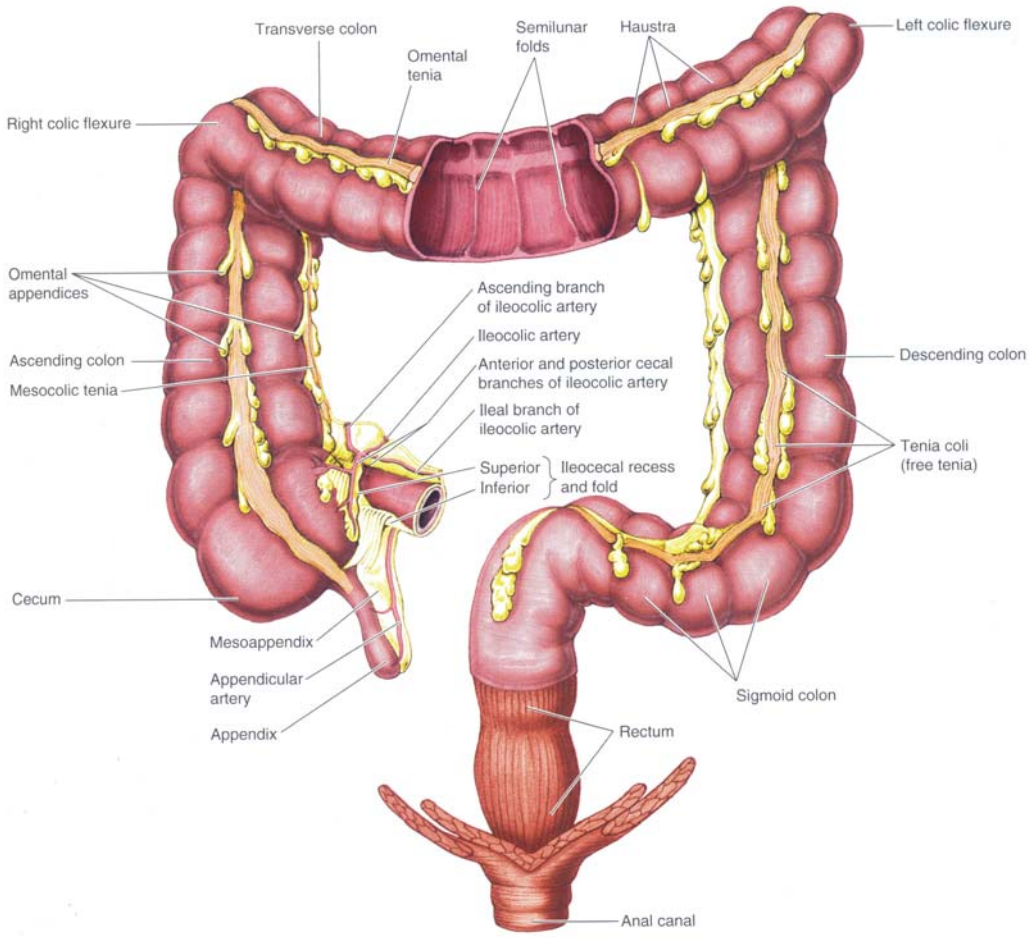
Colorectal surgery had advanced a lot with introduction of endo GIA stapler for sphincter saving procedures.

AIM OF STUDY

The study was undertaken to find out the pattern of

- Incidence - age, sex and site wise
- Risk factors
- Modes of presentation
- Treatment modalities
- Adjuvant therapy settings
- Follow up of colorectal carcinoma in Coimbatore Medical College Hospital, Coimbatore.

ANATOMY OF THE LARGE INTESTINE



SURGICAL ANATOMY OF LARGE INTESTINE COLON and RECTUM

The large intestine extends from end of ileum to anus and comprises of the caecum (with appendix) colon, rectum, anal canal, measuring between 110-170 cm in length[1] (on an average 135 cm long). The caliber is greatest at its commencement at the caecum and gradually diminished as it is traced distally, but again becomes more dilated in the lowermost part of rectum just above the collapsed anal canal.

EMBRYOLOGY

The large intestine develops form both mid gut and hind gut. Midgut portion extends form caecum to the proximal 2/3 of transverse colon supplied by superior mesenteric artery.

Hindgut – from distal 1/3 rd of transverse colon to proximal anus supplied by inferior mesenteric artery. The distal anal canal is ectodermal in origin and supplied by internal pudendal vessels.

Large guts starts developing by fifth week of gestation and is completed by eight week of gestation when the anal membrane ruptures. During sixth week, migration and midgut rotation occurs

over 4 weeks assuming final anatomic position by 10th week of gestation.

ANATOMY

The Caecum

The caecum lies in the right iliac fossa, app 6 cm in length and 7.5 cm in breadth[2]. Proximally becomes ascending colon at its junction with terminal ileum guided by a valve which prevents reflux (contains muscle).

It lies on iliac and psoas muscle and on genitofemoral, lateral cutaneous nerve of thigh. Its exact position is variable, may extend into true pelvis. It is almost completely enveloped by peritoneum but devoid of mesentery[3] and often it is attached to iliac fossa medially and laterally.

THE ASCENDING COLON

It varies from 10-20 cm (avg. 15 cm app). It lies on iliacus - muscle, iliac crest, quadratus lumborum and crossing lateral cutaneous nerve of thigh, ilioinguinal and iliohypogastric nerve. It is usually covered with peritoneum on all 3 sides except posteriorly where it is fixed to post abdominal wall. Sometimes it may be fixed by a short mesentery. It ends at hepatic flexure where it turns left on the lower portion of right kidney.

THE HEPATIC FLEXURE

At this point the ascending colon turns sharply medially and slightly forwards and downwards just below the right lobe of liver and overlapped by it and posteriorly lies on lower aspect of right kidney.

THE TRANSVERSE COLON

It is the longest of all, varying from 40 cm – 70 cm in length extending from hepatic flexure to splenic flexure forming a dependent loop between the points. It is suspended by transverse mesocolon which is attached to descending part of duodenum, lower aspect of body of pancreas and anterior surface of left kidney.

It contains middle colic vessels and branches of left colic artery, right colic artery and lymphatics. Its posterior relations from right to left are anterior surface of descending duodenum, small intestine, part of left kidney. Just below the spleen it turns down to form splenic flexure.

THE SPLENIC FLEXURE

This flexure lies at the junction of the transverse colon and the descending colon. Here the colon bends downwards and backwards. This flexure lies behind the stomach, and below the anterior end of the spleen on the lower part of the left kidney and diaphragm. The flexure is attached to the diaphram by phrenico colic ligament.

THE DESCENDING COLON

It extends from splenic flexure to rim of true pelvis close to inguinal ligament from where it continues as sigmoid colon measuring 25 cm. Usually it is retroperitoneal. It rests on the same muscle and related to the same nerve as ascending colon.

At anterior superior iliac spine it turns medially, superior to inguinal ligament and lies on femoral nerve, psos muscle, genital vessels, becomes sigmoid colon anterior to external iliac vessels.

THE SIGMOID COLON

It is the most variable part in length (40 - 80 cm) and mobility. It extends upto rim of true pelvis where it becomes the rectum and is suspended by sigmoid mesocolon a long mesentery with short base.

THE RECTUM

It lies in the true pelvis measuring about 12-15 cm with a diameter of 4 cm when empty. It is dilated in the lower part to form ampulla of rectum.

If follows curve of sacrum and coccyx runs anteriorly, inferiorly to central perineal tendon lies on levator ani muscles, anococcygeal ligament. It ends posterior to central perineal tendon and to the apex of prostate in male by turning posteriorly and inferiorly as anal canal.

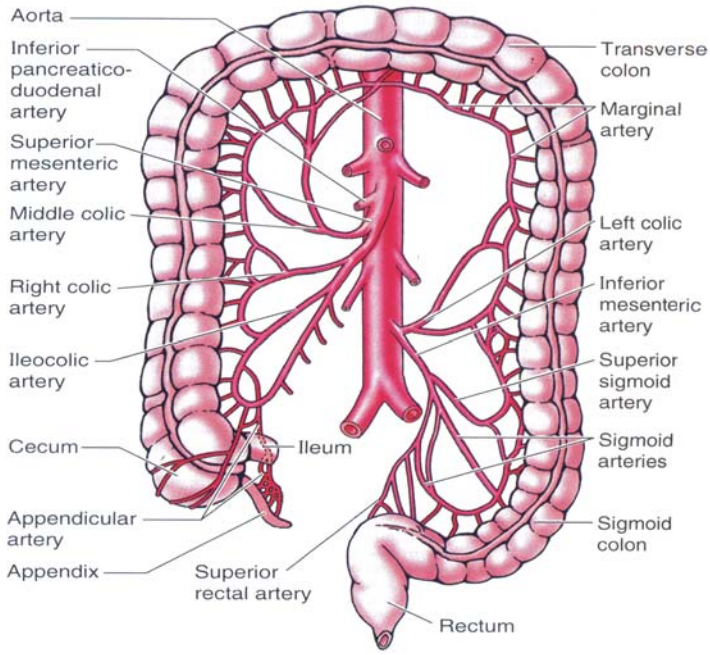
If follows the curve of sacrum, coccyx in saggital plane. In coronal plane it is 'S' shaped giving rise to prominent folds in the lumen known as Houston's valves. The relationship of pelvic peritoneum to rectum is of considerable surgical importance. The upper third has a complete peritoneal investment except for a thin strip posteriorly where peritoneum is reflected as the two leaves of thick mesorectum. As rectum descends into pelvis, the uncovered portion becomes wider until only anterior aspect has a peritoneal coat in middle 1/3 of rectum. This peritoneum gets reflected forward in the bottom of rectovesical pouch or rectouterine pouch leaving lower third of rectum extra peritoneal. Posteriorly the pelvic fascia is thickened to form fascia of Waldeyer separating rectum from sacrum, coccyx, blood vessels, and nerves. Anteriorly separated by the fascial layer known as Denon villier's fascia.

The upper 2/3 of rectum is separated from pelvic fascia by posterior cushion of areola tissue which becomes circumferential below rectovesical / rectourterine pouch carrying blood vessels and its lymphatics known as mesorectum.

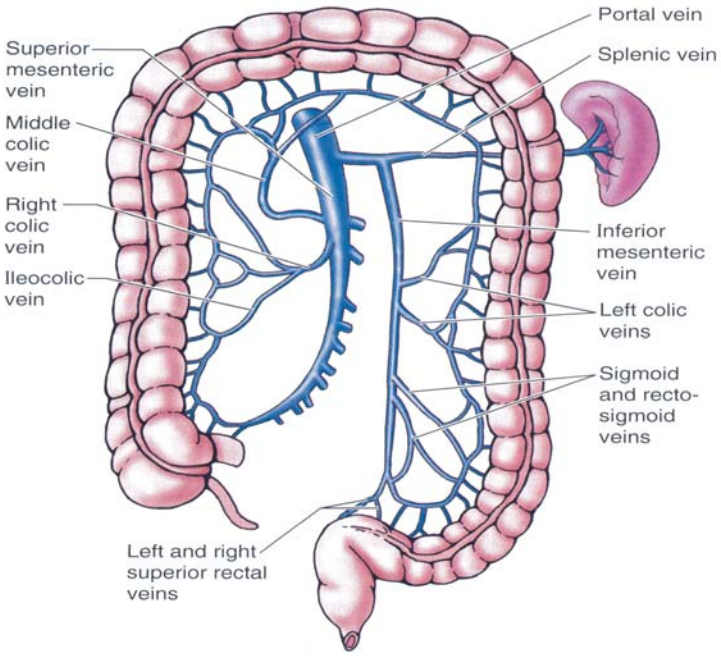
THE ANAL CANAL

It is 3-4 cm long extends from the anorectal junction to the anus. It is directed downwards and backwards and surrounded by

BLOOD VESSELS OF THE LARGE INTESTINE



(A) ARTERIES



(B) VEINS

sphincters which keep the lumen closed in the form of an antero posterior slit, posteriorly anococcygeal ligament separate it from coccyx, while anteriorly perineal body separates it from membranous urethra, penile bulb or lower vagina laterally it is releated to ischiorectal fossa. Its whole length surrounded by sphincters which keeps it closed.

The mucosa of canal consists of an upper mucosal and lower cutaneous part, the junction being marked by line of anal valves about 2 cm from anal orifice known as Dentate line or Pectinate line.

BLOOD SUPPLY AND LYMPHATIC DRAINAGE

These two are important subjects in relation to malignancy and its treatment.

BLOOD SUPPLY

The main arteries supplying the colon, rectum are superior mesenteric artery, inferior mesenteric artery, middle, inferior rectal arteries. The caecum, ascending colon, hepatic flexure and proximal two thirds of transverse colon derives blood supply from superior mesenteric artery originating from aorta at L2 level via ileo colic, (R) colic, middle colic vessels. The distal third of transverse colon, splenic flexure, descending colon, sigmoid and upper third of rectum via inferior mesenteric artery arising from aorta at L3 level via left

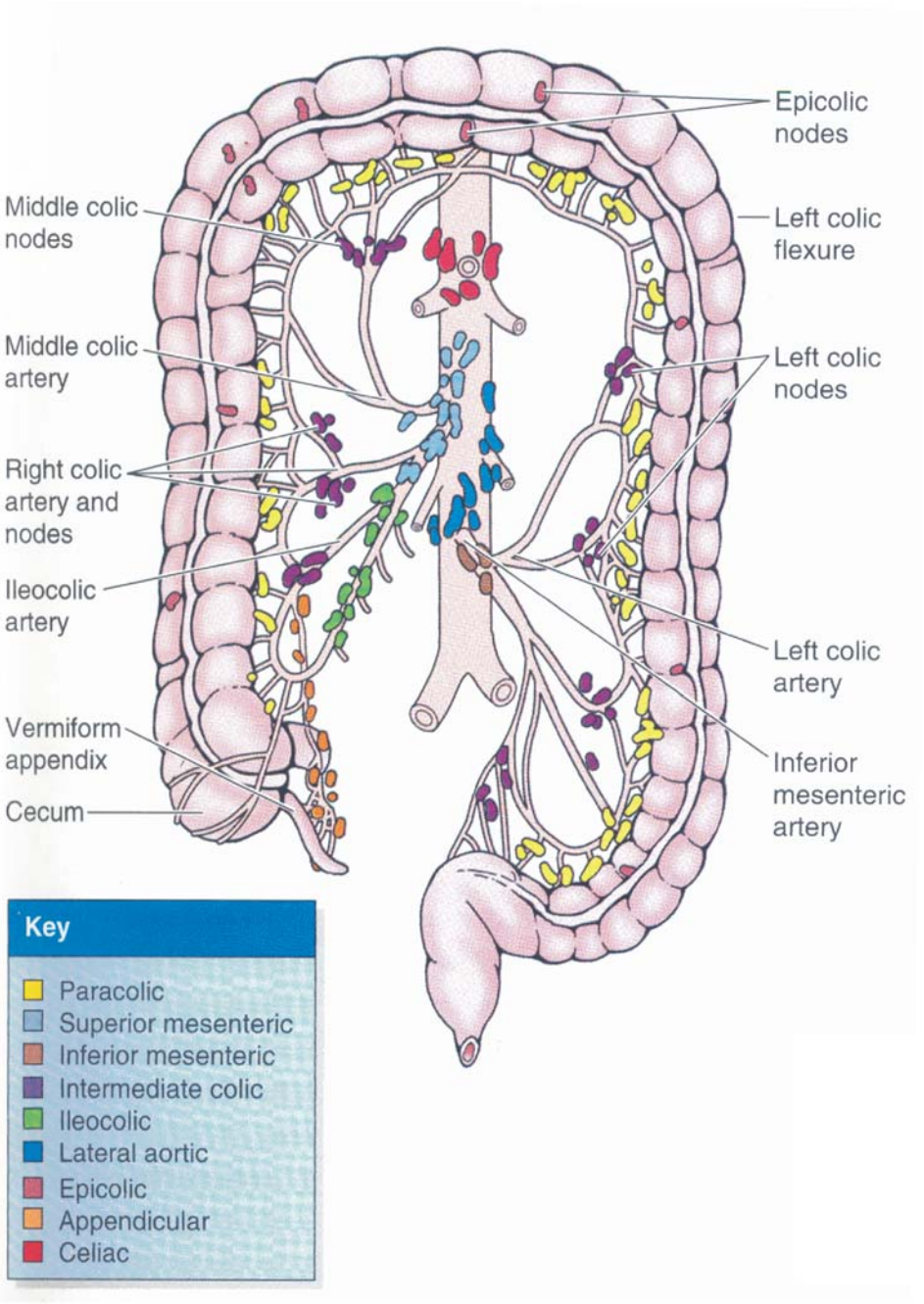
colic, sigmoid branches and superior rectal artery. The distal two thirds of rectum, anal canal get blood supply via middle, inferior rectal artery of internal iliac artery.

The main colic arteries proceed to colon and bifurcate to form branches which unite to form arcades an inch or so from mesenteric border, so that a continuous chain of communicating vessel is formed. This is the marginal artery from which the ultimate branches to the colon, the vasarecti are distributed.

These branches ramify between and supply muscular layers, divide into small submucosal rami and enter the mucosa. The marginal artery is responsible for bringing the area of supply of the superior mesenteric artery into communication with that of inferior mesenteric by connecting the descending branch of the middle colic with the ascending branch of the left colic by means of long anastomosis of colon.

The venous drainage follows its arterial blood supply and empties into portal venous system. The inferior mesenteric vein diverges from artery and passes behind pancreas to drain into splenic vein.

LYMPHATICS OF THE LARGE INTESTINE



LYMPHATIC DRAINAGE

The entire colon and rectum are drained by a large number of lymph nodes numbering 70-100 which are present as a series draining into a principal nodal group.

A. INTRAMURAL LYMPHATICS

Throughout colon and rectum, continuous lymphatic plexus in the submucous and subserous layers of the bowel wall are inter connected and drain into extramural lymphatics.

B. EXTRAMURAL LYMPHATICS OF COLON

These consists of lymphatics channel and glands which are divided into 4 groups.

Epicolic – Minute nodes on colonic wall, sometimes in appendices epiploicae

paracolic – Along the medial borders of ascending colon, descending colon and mesenteric borders of others.

Intermediate colic – Nodes like along right, middle, left ileocolic arteries

Preterminal colic – Nodes adjoining the main trunks of superior inferior mesenteric arteries near their corresponding pre-aortic nodes. These nodes end in pre aortic nodes, which drain into para aortic

groups and hence via efferent channels to thoracic duct into internal jugular vein.

C. EXTRAMURAL LYMPHATCS OF RECTUM

Likewise lymphatics drain in to pararectal group in the wall of rectum, then to intermediate group around main arteries and then to nodes near origin of main vessel.

1. Lymphatics from more than upper half of rectum drain along the superior haemorrhoidal and inferior mesenteric vessel into aortic gland after passing through the para rectal and sigmoid nodes.

2. Laterally along middle haemorhoidal vessels on either side to ischiorectal fossa and thence to internal iliac glands via inferior rectal and internal pudental vessels (above mucocutaneous junction).

3. Lymphatics of anal canal, below Dentate line descend to medial superficial inguinal nodes.

SURGICAL PHYSIOLOGY OF COLON, RECTUM

In man, the large intestine receives the ileal contents, absorbs water and electrolytes and acts as a reservoir for the faecal matter until it is suitable to be discharged through the anus. It was calculated by Smidday et al (1960) that about 800-1000 ml of fluid enters the large intestine each day and 150 ml of this is passed in the feces. Complete loss of colonic and rectal function occurs during ileostomy and total proctocolectomy procedures. The discharge initially high, slowly diminishes as the terminal ileum adapts taking over absorptive function of colon. The importance of terminal 30 cm of ileum was emphasized by Lillehei and Wangensteen (1956) urging conservation if possible[4].

When ileocaecal valve is removed in right hemicolectomy bowel function is altered to give an increased stool frequency upto 4 times a day. This is due to colonic reflux with bacterial colonization of small bowel and loss of regulating valve. After left hemicolectomy only a slight increase in stool frequency occurs.

INCIDENCE / EPIDEMOLOGY

It is a dynamically changing disease entity due to multifactorial reasons. It is predominantly a tumour of old age > 50 yrs and can occur also in young individuals (genetic inheritance).

90% of carcinoma occurs in people more than 50 yrs old. There is a definitive male preponderance (more in rectal than colon carcinoma) averaging 1.3 - 1.8 : 6 sex ratio.

The incidence of colo rectal cancer is much higher in western countries suggesting environmental and genetic factors. The incidence is increasing in our country over the last few years possibly related to changing dietary, social habits. The age standardized rates of colorectal cancer in India is 4.2 and 3.2 per lakh for males and females[5].

It is observed from earlier statistics Smiddy, Goligher (1967) that recto sigmoid accounts for more than half cases of colorectal cancer. Now there is a progressive trend towards disease of right colon[6].

Carcinoma of rectum accounts for nearly one third of all cancers, followed by carcinoma of sigmoid colon, cancer caecum and recto sigmoid junction - followed by others. In order of decreasing

frequency in others are transverse colon, ascending colon, descending colon, splenic flexure and hepatic flexure (Bailey & Love)[7].

In rectum, great controversy exists so as to the distribution of cancer. According to statistics 36% growth upper third 29.8 % middle third, 38% lower third occurs.

ETIOPATHOGENESIS

The exact cause of colorectal cancer is not known precisely, with recent work providing that a complex interaction between genetic makeup and the environment in which he resides determining the incidence. Majority of neoplasms are adeno carcinomas, with malignant melanoma, squamous cells carcinoma being rare variants in anal canal.

ETIOLOGY

Genetic Predisposition

Approximately 20% of colorectal cancer is familial[8]. These include familial adenomatous polyposis, hereditary non polyposis colon cancer, Peutz Jeghers syndrome, Juvenile polyposis.

Hereditary non polyposis colon cancer

Two distinct clinical presentation were made out.

Lynch Syndrome I - Site specific proximal colon cancer in the family

Lynch Syndrome II - Characterized by the development of colorectal endometrial, gastric, upper urinary tract, ovarian and other malignancies.

Both groups are defined using Amsterdam criteria. These families found to have microsatellite instability in genes due to mutation in mismatch repair genes[9].

Familial adenomatous polyposis

It is an autosomal dominant syndrome, diagnosed when a patient had more than 100 adenomatous polyps in colon or with a member of an FAP family ahs any number of colonic adenomas detected. The basic defect is due to mutation in APC gene located in chromosome 5 q21 locus[10]. All patients with this defect will develop colonic cancer if left untreated, hence recommendation state periodic colonoscopic examination and prophylactic polypectomy with HPE to rule out carcinoma or prophylactic proctocolectomy. One marker is congenital hypertrophy of retinal pigmented epithlium seen in 70-80% by ophthalmoscopy.

Variants of FAP are

1. Attenuated adnomatous polyposis coli. The patient have less number of polyps with same high risk of malignancy.

2. Hereditary flat adenoma syndrome develop small adenoma, less than 100, frequently dysplatic prone for malignant change.

Others in spectrum of hereditary polyposis syndrome are

A. Gardner's syndrome – Colonic polyposis, epidermal inclusion cyst, osteomas of bone, upper GI tumours.

B. Turcot's syndrome – Colonic polyps with Brain Tumours (medulla blastoma)

Environmental Factors

Diet

Dietary fat is considered to be an important risk factor for colorectal cancer. Saturated fats are more carcinogenic than unsaturated fat[11].

Other compounds suggested to be carcinogenic are fecapentanes produced by gutflora, 3 ketosteriods metabolic product of cholesterol, pyrrolysis product formed by smoking or deep frying meat products, rice, etc. Even increased bile acids are thought to be carcinogenic.

Diet containing high fibre, vegetables and fruits are protective against colorectal cancer[12].

This dietary role is proved by people who immigrate from low risk regions to high risk region acquiring high risk in a generation time.

Carcinogens

No clear relationship has been established between specific carcinogens and colorectal cancer. Potential agents under study include Bile acids, food additives, alcohol, cigarette smoking, ionizing radiation, oxygen free radicals may serve as promoter or stimulants to alter gene development.

Pre malignant Conditions

Ulcerative Colitis

The absolute risk of cancer in ulcerative colitis is 5 to 10% after 20 years of disease. Dysplasia is a precursor of cancer[13].

Crohn's Disease

Overall incidence of cancer is 7% over 20 yrs. Risk of cancer is high in bypassed segments, in fibrotic narrowing and sites of stricturoplasty.

Previous malignant Disease

Patient who underwent treatment for cancer large bowel have three fold increased risk of developing colorectal malignancy.

Polyps

Adenomatous polyps more than 2 cm in size, patient with multiple polyps, villous adenomas when compared to tubular ones have increased incidence.

Influence of hormones and growth factors

The lining of colon is exposed to a variety of endogenous substances exerting tropic effects on mucosa, gastrin appear to be most directly related to colonic carcinogenesis. Elevated levels demonstrated in patients with colorectal cancer. Other growth factors associated are transforming growth factor bombesin, IGF.

Others

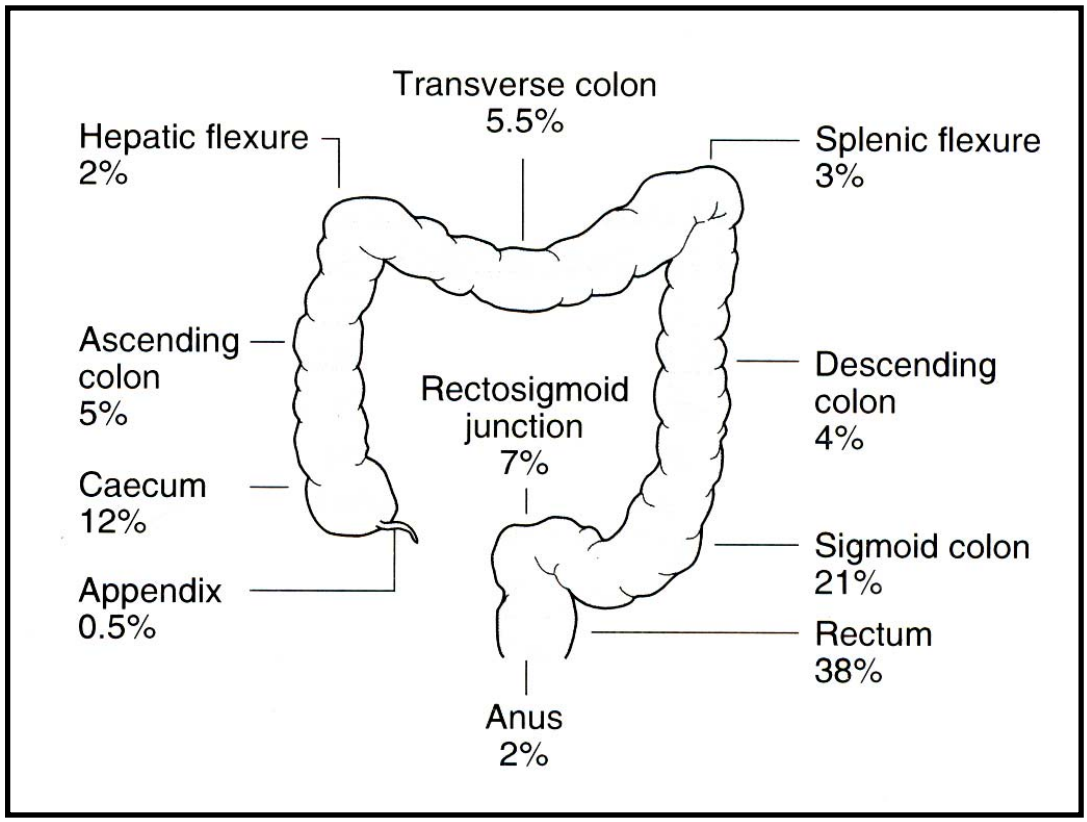
Pelvic Radiation

Supportive, but inconclusive evidence found between radiation and colorectal cancer. Over all risk is very small.

Previous Non Cancer Surgery

Cholecystectomy, uretero sigmoidostomy patients have increased incidence.

DISTRIBUTION OF COLO RECTAL CANCER BY SITE



PATHOLOGY AND SPREAD

In initial stages, cancer of large intestine takes the form of localized area of thickening of the normal mucosa or a hard nodule in a preexisting adenoma or villous papilloma.

There are 4 distinct macroscopic types.

A. Polypoid or cauliflower growth (fungating / Exophytic)

This produces a large fungating mass which projects into the lumen of the bowel and is not usually associated with much infiltration of the intestinal wall more commonly seen in proximal colon, ascending colon, caecum, etc.,

B. Annular or constricting or circumferential growth

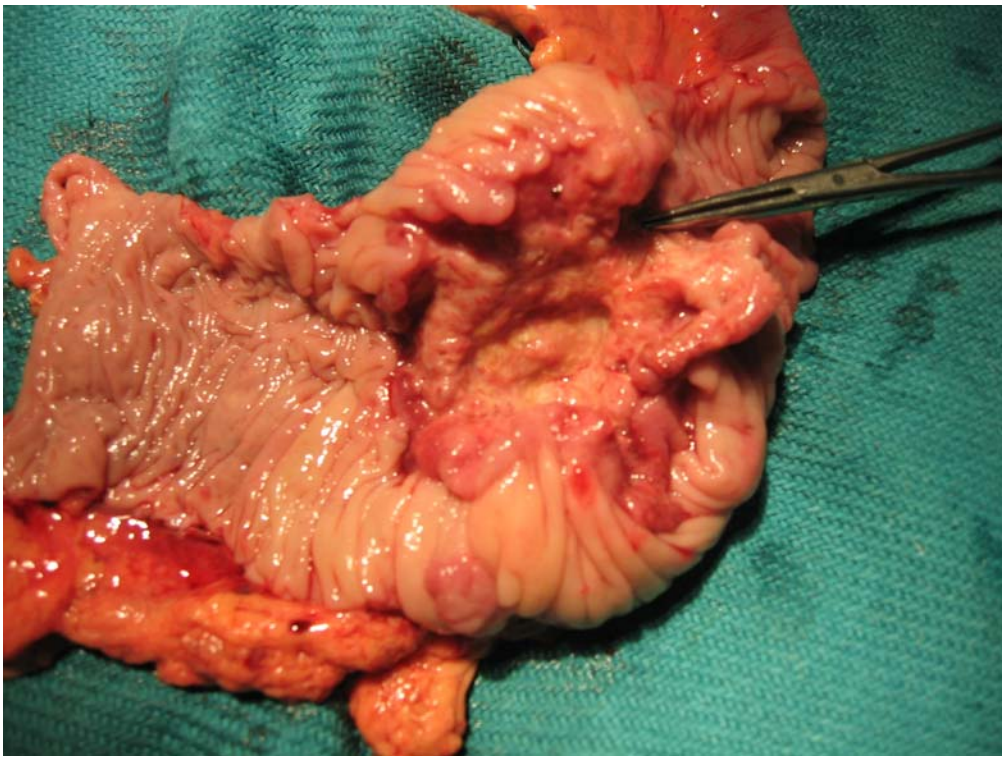
These lesion extends around bowel wall and the bowel looks as if deeply constricted by a string around it more commonly seen in carcinoma of descending and sigmoid colon.

C. Ulcerating Growth

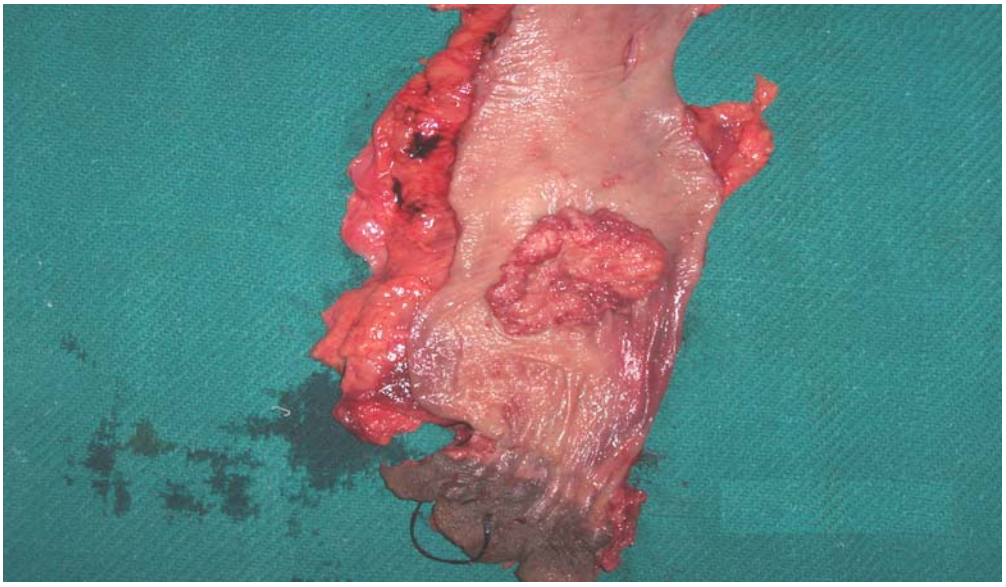
Presents as a typical malignant ulcer and this infiltrates the bowel wall producing deformity and narrowing of the lumen.

D. Diffusely infiltrating growth

This corresponds to linitis plastica of stomach and produces thicknening of the intestinal wall usually extending for at least 2-3”



ADENOCARCINOMA – CAECUM



ADENOCARCINOMA - RECTUM

and for more part covered with intact mucosa. More common in the left side growths.

While all consider the above types as the 4 macroscopic appearances, Duke classified adenocarcinomas which produces abundant mucin as colloid carcinoma.

E. Colloid Carcinoma

This forms a bulky growth with gelatinous appearance and may or may not be associated with ulceration and infiltration.

HISTOLOGIC TYPES

The most common type is adenocarcinoma, the other types are:

Mucinous adenocarcinoma

Signetring cell adenocarcinoma

Squamous cell carcinoma

Adenosquamous carcinoma

Undifferentiated carcinoma

Other types are Carcinoids tumours

Non epithelial tumours

Degree of Differentiation

In general papilliferous growth tend to be better differentiated than the ulcerating or infiltrating type.

BRODERS GRADING[14]

Broders designated adenocarcinoma into 4 grades based on percentage of differentiated tumour cells.

Grade I : Well differentials tumours, closely resembling an adenoma

Grade II : Tumour cells more crowded together but still arranged in fairly regular pattern

Grade III : Less differentiated and arranged in irregularly folded rings

Grade IV : Anaplastic cells which did not form glandular structures at all.

Mucoid tumours vary considerably and were graded separately by Duke

DUKE'S GRADING

Duke considered arrangement of cell and evolved into new three grade system.

Grade I : Well differented – well formed tubules least nuclear pleomorphism and mitosis.

Grade II : Moderately differentiated.

Grade III : Least differentiated – occasional glandular structure more Pleomorphic cells large number of mitosis

SPREAD OF COLORECTAL CANCER

Most of our knowledge of spread was due to studies by Dukes (1930). Gordon – Watson.

A. LOCAL INVASION

First, invasion after initial mucosal growth is to protrude into the lumen. Lateral invasion was more in transverse direction leading to circumferential growth[15]. Mural penetration leads to peritoneal seedling. Additional spread is via perineural spaces with invasion reaching as far as 10 cm from the primary tumour.

B. LYMPHATIC EXTENSION

Lymph nodal metastasis occurred only after tumours has penetrated into perirectal / colonic tissues. Lymphatics spread occurs in an orderly fashion through upwards, laterally and downwards direction[16].

Retrograde spread occurs on blockage of central nodes. The risk of lymphatics spread increases with increasing tumour grade. As spread follows the course of blood vessels supplying the carcinomatous region, appropriate fields of excision is worked out by reference to arterial supply.

C. HAEMATOGENOUS SPREAD

The liver is the primary site of haematogenous spread followed by lungs in colonic cancer. As venous drainage of rectum is via dual systems. The liver contains secondary deposits in about one-third to one half of fatal cases liver[17] and lung are involved primarily depending on site of tumour origin in rectum.

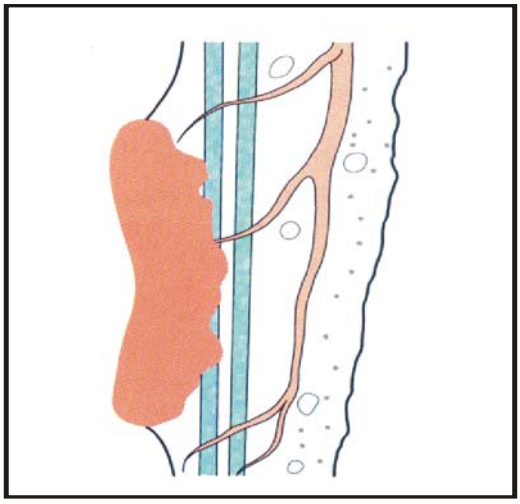
Batson's vertebral venous plexuses represent another way of blood spread of metastasis to bone and CNS.

D. IMPLANTATION

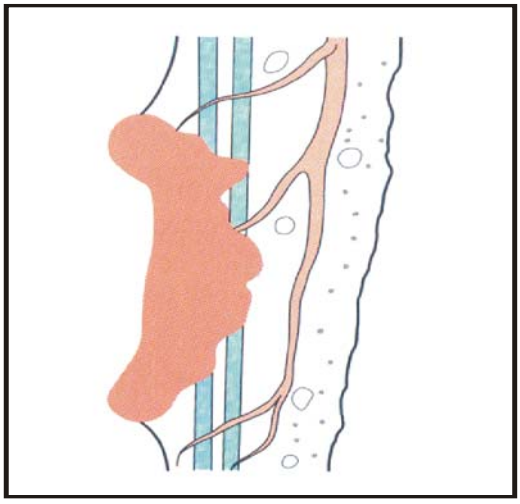
It refers to release of tumour cells from primary site and their deposition on another surface. It can occur transluminally, transperitoneally after serosal invasion and during surgical manipulation diminishing curative resection and increasing local failure rate, and distant deposits.

DUKE'S STAGING

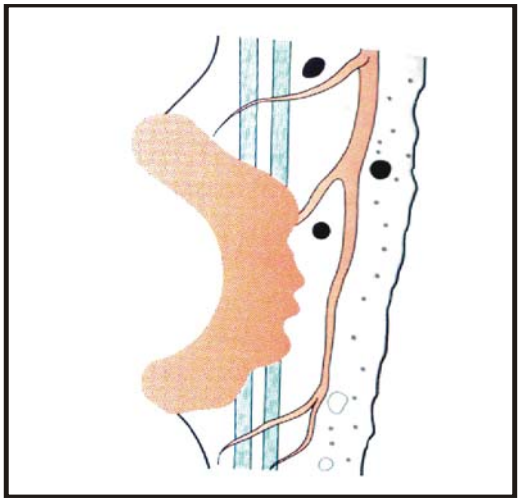
STAGE A



STAGE B



STAGE C



STAGING AND CLASSIFICATION

Duke presented the first original staging for rectal carcinoma which is widely used now to stage both colorectal tumours.

DUKE STAGING (1930)

A – Growth limited to colonic / rectal wall but not through it

B – Growth penetrating through Bowel Wall

C – Involvement of lymph node regardless of extent of Bowel wall penetration

1935 Modification

A, B as same.

C1 - Locally positive Nodes

C2 - Positive nodes up to the point of ligature

KIRKLIN MODIFICATION (1941)

A - Tumour limited to mucosa, submucosa only

Duke A

B1 - Tumour infiltration into, but not through Muscularis Propria

B2 - Tumour infiltration through – muscularis propria

Duke C - Remain same

D - Defined as disease beyond limit of surgical Resection (Turn bull addition)

ASTLER and COLLER MODIFICATION[18]

A - Limited to Mucosa

B1 - Extending into, but not through in propria involved nodes.

B2 - Extending into and through muscularis propria, with uninvolved nodes.

C1 - Extending into, but not through muscularis propria involved nodes.

C2 - Extending through M. Propria with involved nodes

This staging allowed separation of wall penetration and nodal status.

TNM CLASSIFICATION

The AJCC has recommended TNM classification which covers all possible presentations.

PRIMARY TUMOUR

Tx : Tumour cannot be assessed

To : No evidence of tumour in resected specimen

Tis : Carcinoma in situ

T1 : Tumour invade submucosa

T2 : Invades muscularis propria

T3-4 : Depends on presence / absence of serosa

Serosa Present :

T3 : Invades through muscularis propria into the serosa (but not through)

Pericolic fats within the leaves of mesentery

T4 : Invades through serosa into free peritoneal cavity or into a contingous organ

Serosa Absent :

T3 : Invades through muscularis propria

T4 : Invades other organs

REGIONAL LYMPH NODES

Nx : Nodes cannot be assessed

No : No regional node metastasis

N1 : 1-3 positive nodes (Pericolic / perirectal)

N2 : 4 or more positive nodes

N3 : Central nodes positive

DISTANT METASTASES

Mx : Presence of distant metastases cannot be assessed

Mo : No Distant metastases

M1 : Distant metastases present

STAGING[19] :

Stage	T	N	M
0	T_{1s}	N_0	M_0
I	T_{1-2}	N_0	M_0
II A	T_3	N_0	M_0
II B	T_4	N_0	M_0
III A	T_{1-2}	N_1	M_0
III B	T_{3-4}	N_1	M_0
III C	Any- T	N_2	M_0
IV	Any – T	Any - N	M_1

CLINICAL FEATURES

There are 3 main ways in which carcinoma of large intestine may present.

A. As non emergency cases with insidiously developing chronic symptoms chiefly affecting bowel function and general health.
B. As emergencies with perforation / obstruction of colon with or without peritonitis.
C. Non specific symptoms

The common symptoms comprise the following

- Altered bowel habits in the form of alternating constipation and Diarrhea, tenesmus etc.
- Sense of incomplete evacuation
- Bleeding per rectum
- Mucus per rectum
- Mass per abdomen
- History of piles / haemorrhoids
- Abdominal pain / dyspepsia
- Loss of weight, asthenia, impairment of general health
- Loss of appetite
- Flatulent dyspepsia

- Acute on chronic bowel obstruction
- Bowel perforation / peritonitis

Fairly definite correlation between site, type of growth and symptomatology occurs. Carcinoma of left colon and rectosigmoid present early due to stenosus / circular growth whereas right colon growths present late. Local spread may present with related symptoms like rectovaginal fistula, recto vesical fistula, uterine obstruction, hemorrhoids etc.,

Carcinoma of caecum and ascending colon

Mostly present with anemia, severe and unyielding to treatment. A palpable tumour may be present. Sometimes discovered unexpectedly at operation for acute appendicitis for an appendicular abscess failing to resolve. Sometimes carcinoma of caecum can be the apex of an intussusception presenting with intermittent obstruction.

Carcinoma of transverse colon

It may be mistaken for carcinoma of stomach due to position of tumour together with anemia and lassitude.

Carcinoma of left side of colon

Majority of tumours occur in this location are usually of stenosing, annular, ulcerative type. The main symptoms are those of increasing intestinal obstruction. Colicky or constant aching pain may

be the only symptom. Alteration in bowel habits, palpable lump, abdominal distension are the other symptoms.

Carcinoma of rectosigmoid

It presents usually with bleeding per rectum, sense of incomplete defecation, tenesmus and alteration in bowel habits. Subacute intestinal obstruction, may induce colicky pain in abdomen. Pain in the back sciatica indicate local sacral plexus involvement. History of piles may be the presenting complaint. Symptoms pertaining to local or distant spreading may be the initial presentation in some. May present as fistula in ano, single or multiple discharging pus.

Carcinoma of Anal Canal

Commonly presenting early with bleeding P/R, pain per anal region, mass per anal region. Patient may present with constipation, fistula in ano, obstruction, abdominal distension.

Colorectal carcinoma presenting with acute obstruction

About 1/5 of patient complete obstruction occurs either an aggravation of chronic affairs as acute on chronic obstruction or may present one day suddenly as pain, distension of abdomen due to the narrowed lumen plugged by a fecolith, hard stools, undigestable fibres.

Chronic obstruction is more commonly encountered with left colonic carcinoma rather than right sided lesions. Constricting stenosing types seen more on left side and the faecal nature produces greater occurrence of acute obstruction.

Symptoms

Complete obstruction of large bowel is often entirely insidious in onset. If acute on chronic variety with development of acute obstruction, the patient finds he is constipated without passing motion for 2-3 days despite use of apperients. He is not able to get rid of flatus and with progressively increasing abdominal distension. This state may be prolonged with slow increase in abdominal discomfort and distension over a period of 6-7 days before finally presenting to hospital.

Carcinoma colon with Bowel Perforation

Colonic perforation occurs in 3-8 % of patients with colo rectal cancer. It's often the result of obstruction followed by perforation. The common belief was that perforation takes place in a stercoral ulcer usually in the caecum and less commonly closer to growth. But in a series of 115 cases of carcinoma of large intestine presenting with perforation only 20 cases of this type were found, while majority was

proximal to an obstructing cancer producing localized abscess or generalized peritonitis.

SYMPTOMS

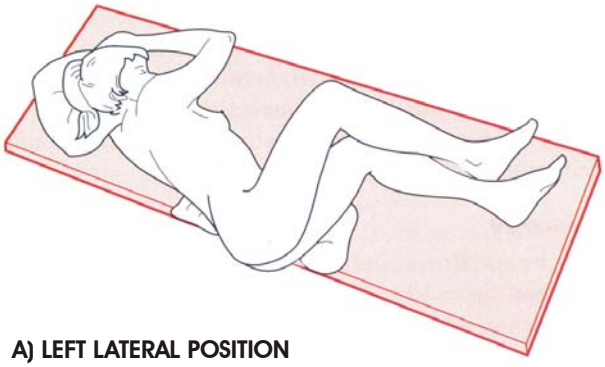
Obstructed cases with perforation

These patients are usually desperately ill with abdominal distention, diffuse tenderness, vomiting, gross dehydration and electrolyte imbalance. The underlying carcinoma may / may not be palpated or suspected from clinical obstruction. In majority of cases, unless, supportive measures are done rapidly to stabilize the patient, patient condition rapidly deteriorates to end fatally.

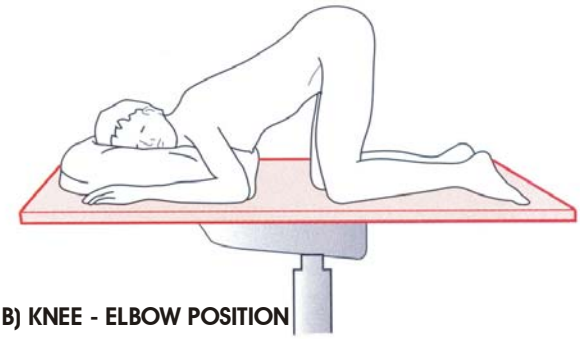
Unobstructed cases with perforation

Most of these patients are also gravely ill and shows signs of general peritonitis, but owing to the absence of previous obstruction, contamination is less. In some, perforation results in localized peritonitis, abscess formation, producing diagnostic confusion.

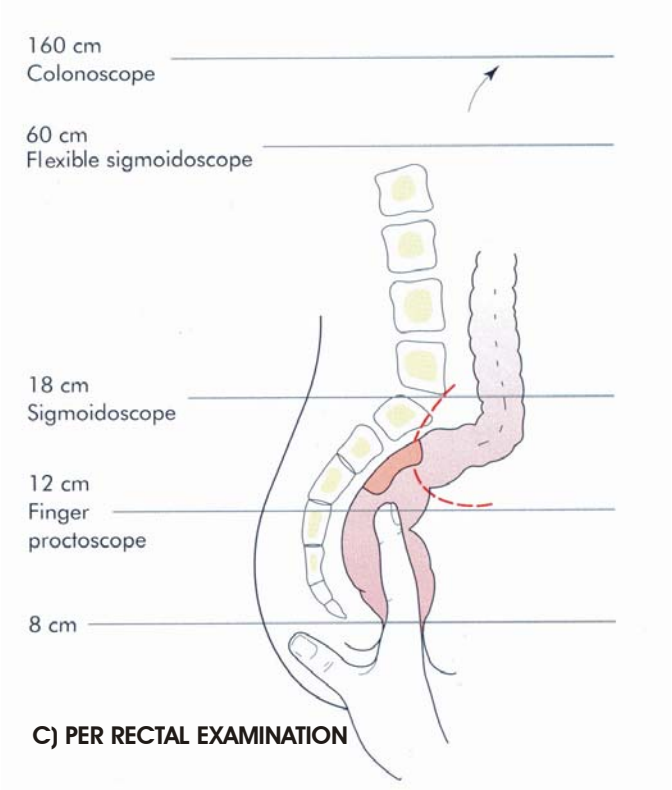
If a caecal growth perforate and subsequent development of subcutaneous abscess, the presentation may mimic an appendicitis or abscess formation.



A) LEFT LATERAL POSITION



B) KNEE - ELBOW POSITION



C) PER RECTAL EXAMINATION

INVESTIGATIONS

Routine laboratory investigation to be done includes complete hemogram, B. Urea, B.Sugar, Sr.Creatinine, Urine R/E, E.C.G., Chest X ray, X-ray abdomen, including liver function test to ascertain general condition of the patient and treatment of these abnormalities to allow treatment of cancer.

Definitive test for detection / diagnosis of colorectal malignancy

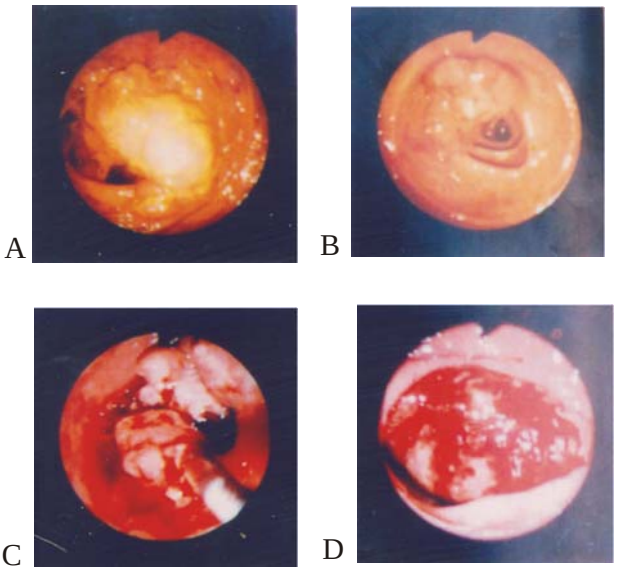
Test for occult blood

Guaiac test – detects 20 mg hemoglobin per gram or 20 ml blood / day. High false positive results occur due to presence of other oxidizing agents.

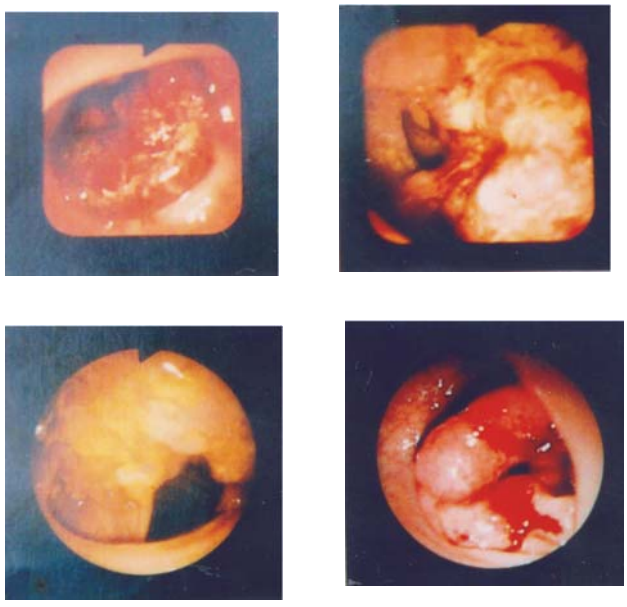
Immuno fluorescent test – detects conversion of hemoglobin to fluorescent porphyrins, detects 5-10 mg hemoglobin / gm of stool. False positive prohibitively high.

Rectal examination

Reaches approximately 8 cm above dentate line upto 75% of all rectal tumours and 35% of all large bowel tumours can be palpated.



COLONOSCOPIC VIEW - POLYPOID CARCINOMA
SIGMOID COLON (A), TRANSVERSE COLON (B) BIOPSY TAKEN (C)
HAEMORRHAGIC POLYPOID CARCINOMA (D)



ADVANCED CARCINOMA - COLONOSCOPIC APPEARANCE

Proctosigmoidoscopy

Rigid one is 2 cm in diameter 25 cm long, reaching 20-25 cm from dentate line, detect 20-25% of tumours. It is more uncomfortable and the examination is limited to the rectum.

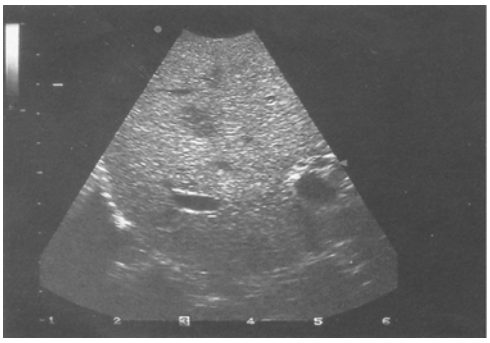
Flexible sigmoidoscopy

It measures 60 cm in length reaching upto splenic flexure and identify 50% of tumours can be done after 2-3 prep enemas, used only as a diagnostic tool with biopsy taking with no therapeutic maneuvers recommended for people over 50 to be years every 3 years in average risk ones.

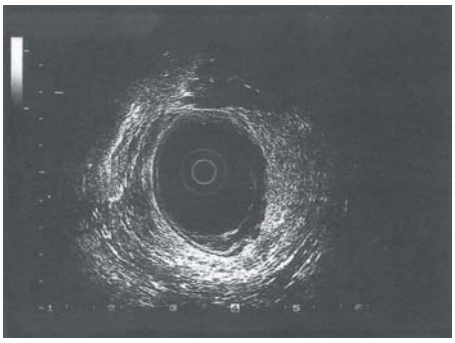
Colonoscopy

Allows visualization of mucosa of entire colon, rectum and usually terminal ileum. Measure about 160 cm in length, it allows biopsy, polypectomy, heamorrhage control, stricture dilatation. Major complication rate < 0.2% with (bleeding, anesthetic complications, perforations) experienced colonoscopists,

Recommended annually or once in 2 yrs in patients with high risk of colorectal malignancy with detection of upto 100% of all tumours by a experienced one.

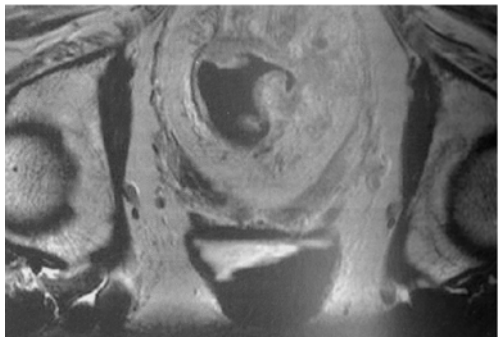


ULTRASOUND APPEARANCE OF
HEPATIC METASTASES



TRANSRECTAL ULTRASOUND APPEARANCE OF RECTAL TUMOR.
T2 TUMOUR SHOWING BREACHING OF MUSCULARIS PROPRIA

BARIUM ENEMA APPEARANCES OF A COLONIC CANCER
WITH TYPICAL ' APPLE CORE' SHOULDERING



MRI SCAN OF PELVIS SHOWING EXTENSIVE
T3 RECTAL CARCINOMA INVADING LEFT SIDE
OF MESORECTUM.

CT SCAN SHOWING MULTIPLE
HEPATIC METASTASES

Plain radiology

Plain abdominal x-ray films are particularly useful in large bowel obstruction showing typical gas pattern.

Contrast studies

The mainstay of radiological investigation is the barium enema. Apart from plain Barium enema. Nowadays double contrast barium enema is used with more accuracy. Water soluble contrast can be substituted for barium in patients suspected of perforation. The cardinal finding is filling defect in the barium shadow.

Imaging techniques

These are important in the evaluation, staging and follow up of colorectal cancer patients.

CT Scan

It allows preoperative evaluation of abdominal cavity to identify metastasis. Integrity of urinary tract and staging the primary tumour lesion esp rectal cancer. Angio CT is 95% sensitive to detect metastasis esp liver. Also 60-80% rectal wall invasion and 75% 1 cm lymph adenopathy can made out by CT Scan.

MRI Scan

New technique for evaluating colorectal cancer phased array pelvic coils used to detect / stage rectal cancer. MRI is more specific

for differentiation of cancer from normal tissue and fibrotic scar. It is also more specific to make out liver metastases esp. resectable ones.

Positron – Emission Tomography

It's still investigational, and may be most helpful in evaluating recurrent tumour in pelvis when dense scar tissue is present, other major advantage is to identify extrahepatic / intraperitoneal disease before surgery.

Abdominal Ultrasound

It is widely used for the detection of hepatic metastases both preoperatively and in follow up and also used for elucidation of abdominal mass lesion.

Trans rectal ultrasound

Also known as endosonography of rectum used to stage rectal cancer USG image allows clear deliniation of rectal wall layers, depth of invasion, adjacent origin involvement.

Positive predictive value T < 1cm - < 50%

> 1 cm – 70%

Endoluminal USG

Colonoscopic USG is available, allowing determination of depth of invasion, attachment to adjacent structures, mesenteric lymph nodal spread. Also used for USG guided LN Biopsy.

Tumour Markers

CEA Assay

In 1965 Gold and Freedman, isolated a specific antigen in adenocarcinoma of the endodermally derived epithelium of the GIT[20]. They were also able to demonstrate the same antigen in embryonic and foetal digestive tract tissue upto the end of the first 6 months of pregnancy circulating antibodies to its were found in the sera of a high proportion of women throughout pregnancy.

Thomson et al (1960) developed RIA capable minute quantities of CEA in serum[21]. Moore et al (1971) further investigated CEA and noted that apart from colorectal carcinoma it was found in carcinoma pancreas and other GIT cancers, bronchogenic carcinoma, alcoholic liver disease and uremia.

The main use of CEA assay will be to monitor patients who had radical surgery for detect recurrence of disease, persistance of the lesion in metastatic form[22]. Other tumour markers include CA 19-9 and CA-50.

Immuno scintigraphy

A recent development in diagnostic tools utilizes the radiolabelled antibody to target tumours for imaging or detection. It is aimed at more specific and sensitive tumour imaging. A glycoprotein

TAG 72 has been found to be useful target antigen in colorectal cancer, the expression of which has been found to be high as 94% in colonic adenocarcinoma.

A study using indium III labeled TAG 72 for imaging colon cancer showed sensitivity of 70%.

Specificity – 90% and accuracy 72%.

BIOPSY

HPE of biopsy obtained preoperatively either by open or via colonoscope or proctosimoidoscope is done to study the type, grade of tumour and other characteristics.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Most of the cases of carcinoma rectum can be detected by a proper rectal examination and this should be mandatory for any patient presenting with suggestive symptoms or a history of piles. Colonic cancers need further investigations described earlier. Faecal occult blood should be done for every patient at high risk. Patients at high risk should be screened by luminal contrast studies.

The investigations available in this hospital apart from routine basic investigations are luminal contrast studies, colonoscopy, USG abdomen and CT scan. In patient who presented as acute emergencies, diagnosis confirmed by exploratory laparotomy.

DIFFERENTIAL DIAGNOSIS

Conditions simulating colonic and rectal carcinoma include ileocaecal tuberculosis, appendicular masses, diverticulitis, polyps etc. Rectal carcinoma should be differentiated from squamous cell carcinoma of anal canal as the treatment is entirely different for this.

TREATMENT

The various modalities of treatment for colorectal carcinomas are :

1. Surgery
2. Radiotherapy
3. Chemotherapy

SURGERY

Surgical resection remains the mainstay of treatment of colorectal malignancies. Due to biological unfavourable response to chemotherapeutic drugs and radiotherapy due to various reasons, surgery is still the best palliative treatment modality.

The objective of surgery in treatment of colonic carcinoma is to remove cancerous segment of bowel, the mesentery containing its lymphatics and any organ directly invaded by the tumour. The way surgical resection is done consists of major steps to give therapeutive curative resection with as little morbidity and morality to the patient and starts preoperatively itself.

Preoperative bowel preparations

As large intestine contain the maximum number of pathogenic bacteria any surgery performed on colon and rectum are classified as

clean contaminated surgical procedures. Steps are taken to reduce bacterial population as much as possible via 2 components.

- Mechanical cleansing
- Antibiotic administration

Mechanical Cleansing

The most commonly used method is dietary restriction to fluids only for 48 hrs before surgery. On the day before surgery, preparation of bowel is by using balanced salt solutions containing purgatives, most commonly mono, dibasic sodium phosphates, polyethylene glycol and fleet's phospho soda with liquids are used.

Antibiotic administration

Mechanical cleansing reduce absolute number of colonic bacteria but wound infection rate is still high (30-60%). Hence administration of antimicrobials by oral, IV routes to reduce colonic bacterial concentrations. Most ideally used ones is IV antibiotics immediately before surgery which reduces postoperative wound infection to about 10%[23].

Surgical Technique

Techniques to minimize tumour spillage, remove adequate bowel length and bowel continuity restorations are as important as surgery itself.

The abdomen is opened and tumour is assessed for resectability by

- Palpating liver for secondaries by visual, palpatory and intropeative USG if available. (Isolated / 3 metastases with in 1 resectable lobe is not a contraindication for curative surgery).
- Palpate peritoneum draining lymph nodes and mobility and fixity of tumour to adjacent organs or abdominal wall structures.

Then intestine to be resected is tied both proximally and distally to prevent intraluminal spread. The major segmental artery supplying the cancerous bowel is ligated, and divided. Any adhesion to adjacent structures are resected in toto if feasible to avoid loss of curative resection (most adhesion, lymphadenopathy may be inflammatory and can only be proved histologically).

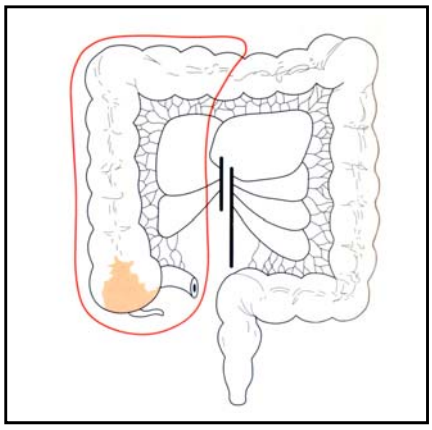
The extend of resection depends upon primary location of the tumour lymphatic metastases, adjacent organ invasion. To get a tumour free margin, usually 2.5 cm margin clearance is ideal, and anastmosis may be end or end to side which should be tension free.

Treatment options depending on Tumour Location

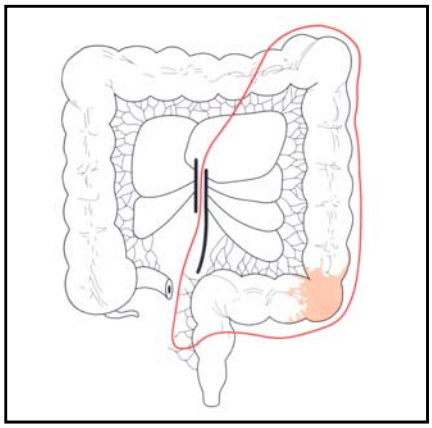
Carcinoma Caecum or Ascending colon :

Treated by a right Hemicolectomy

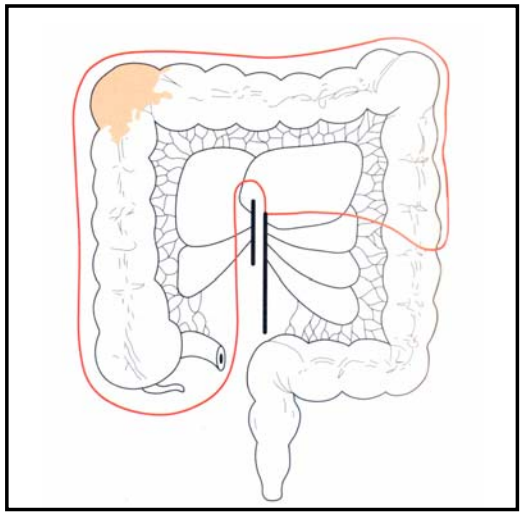
AREA TO BE RESECTED WHEN THE GROWTH IS SITUATED IN THE CAECUM



AREA TO BE RESECTED WHEN THE GROWTH IS SITUATED AT THE HEPATIC FLEXURE



AREA TO BE RESECTED IN THE CASE OF CARCINOMA OF THE PELVIC COLON



Carcinoma Hepatic Flexure

Treated usually by right hemicolectomy or extended right hemicolectomy.

Carcinoma Transverse colon

Depending on position of tumour on transverse colon. Segmental excision of transverse colon. Extended right hemicolectomy may be performed.

Carcinoma splenic flexure

Treated by resection of colon including transverse, descending if needed sigmoid colon with associated lymphatics and mesentery.

Carcinoma descending colon or sigmoid colon

Treated by left hemicolectomy with anastomosis.

Carcinoma Rectum

The surgical treatment of rectal cancer should accomplish complete removal of the cancer and involved regional tissue. Basic principles of cancer surgery is accomplished first and then consideration is given to restoration of intestinal continuity. In surgical treatment procedures, rectal cancers are considered in 3 distinct regions in relation to anal verge (sections of roughly 5 cm intervals). One of the main problems in the treatment of rectal cancer

is the development of local recurrences, the rates vary from 5% to 45%[24].

Upper third lesion

The treatment of distal sigmoid and intra abdominal rectal (rectosigmoid) cancers. Anterior resection, lower anterior, resection is done with restoration of intestinal continuity with sutures / staples[25]. Cure rate and pattern of recurrent cancer similar to proximal colon (Sigmoid colon, descending colon).

Middle third lesion

Can be treated by both abdominal perineal resection or sphincter saving procedures. It depends on tumour location, infiltration, craniocaudal spread with restoration of intestinal continuity in latter procedures.

Lower third lesions

Most of the tumours abdomino perineal resection, rarely some lesion allowing abdomino sacral resection or anterior resection. Selected patients can be treated by restorative protectomy and colonal anastamosis.

Summarizing the various optional modalities for rectal carcinoma are as follows

- ❖ Abdomino perineal resection

- Low anterior resection
    - End to end / side to end
    - Sutures / staples
- Abdomino sacral resection
- Coloanal resection
- Localised procedures[26]
    - Local excision
    - Fulguration
    - Endocavitary irradiation or brachy therapy

Tumour free margins are usually achievable for colon cancer and usually a marging of 5 cm is deemed adequate, since < 2.5% of tumour have intramucosal spread 0 more than 2 cm. During low anterior resection, distal margin of 2 cm is deemed adequate for well differentiated, small non bulky tumour with favorable prognostic features thereby aiding restoration of intestinal continuity.

When synchronous colonic cancers are found at different sites in the colon, the procedure is subtotal colectomy. Resection of contiguous organs for locally invasive tumous is approximately 10% of case. Most commonly involved organs are bladder, ovary, ureter, abdominal wall, less frequently small intestine, spleen, pancreas, stomach and uterus.

ROLE OF LAPAROSCOPY[27]

The oncological safety of laparoscopy is of less concern than the case some years ago. The specter of port site metastasis, once so alarming, has faded. From all of the large scale studies the accumulating evidence seems to point to equivalence in terms of disease specific recurrence and survival between patients treated using conventional and laparascopic techniques.

Usually laparoscopic assisted method are used and fully laparoscopic method are also attempted. They are claimed to facilitate patients convalescence, and reduces stress.

SURGICAL INTERVENTION IN EMERGENCY PRESENTATION

The major emergency presentation is obstruction, and perforation.

Obstruction

The incidence of acute obstruction with carcinoma of the right and transverse colon was 27.4% and with carcinoma of the left colon 40.4% and in the rectum it was only 4.2%. Immediate surgical treatment is necessary. Obstructing right / transverse colon cancer treated by resection and primary anastamosis, while left sided cancer obstruction is a more difficult problem. Appropriate, surgical

procedure depends on location, presentation findings, surgeons experience and judgement. It can be treated in stages with defunctioning colostomy and later curative surgical resection with anastamosis.

Various modalities available

- Primary resection with exterioration of both ends
- Hartmann's procedure
- Primary resection with anastomosis
- Primary resection with anastamosis protected by colostomy or ileostomy
- Subtotal colectomy with ileosigmoidostomy

Perforation

According to statistics, 7% of all cases of carcinoma of the large intestine present with perforation of a bowel and consequent peritonitis. It is usually a life threatening emergency through exploration of peritoneal cavity is mandatory.

Goal of Surgery

Remove diseased, perforated segment as dehiscence rate is very high in a contaminated field. Do anastamosis, protected by proximal colostomy / ileostomy[28]. The diverting stoma closure is done 10 weeks later, the peritoneal contamination irrigated and sucked out.

The survival rate reaches upto 30% of curative resection at 5 yrs in these patients.

ADJUVANT TREATMENT FOR COLORECTAL CANCER

RADIOTHERAPY

The role of RT is well defined in rectal carcinoma than colonic carcinoma. Because colonic carcinoma is intra abdominal, makes radiotherapy less feasible. In rectal cancer local failure with recurrence is the most common presentation making adjuvant radiotherapy worth while.

In Rectal carcinoma, local recurrence in pelvis is due to inability to do wide resection due to narrow confines of pelvis. So radiotherapy can be used in various regimens to prevent local failure.

- ❖ can be used preoperatively
- ❖ sandwich technique
- ❖ post operative irradiation

Usually 4500 – 5000 cGy is delivered over 5-7 week period and little complications results during preoperative use and complications are more after post operative irradiation[29].

Unresectable tumours, patients refusing surgery and barely operable growth may be treated with palliative radiotherapy[30]. Also can be used for palliation of painful recurrences and metastases.

Radiotherapy can be applied using Brachy therapy and Teletherapy. Most commonly used teletherapy are Cobalt therapy and Neutron beam irradiation. Radioactive isotopes are used in brachytherapy with short half lifes.

CHEMOTHERAPY

Chemotherapy is appealing and most effective when tumour burden is minimal / smallest and when the fraction of malignant cells in growth phase is highest. It is most commonly given following the surgical procedures.

Hence maximum response is obtained for postoperative regimen usually for stage III disease with no significant improvement in patient with stage II disease. Most experimented drugs are 5 flurouracil alone or 5 fluroacil and levamisole or 5 Fu and leucovorin combination[31]. New combination regimens are underway and results awaited esp. Methyl – CCNU / vincristine etc.[32]

Currently systematic CT is used to improve long term survival by reducing incidence of distant metastases, to improve local disease control, by combination with external beam radiation, found to have modest survival benefits in stage II, III patients. The most promosing drug is 5 Fu which acts as radio sensitizer in the chemoradiation technique.

IMMUNOTHERAPY

Various agents have been tried to modulate or stimulate innate immune response on enhancement of immune response to destroy, prevent tumour progression, implantation or metastasis. No encouraging results are reported after randomized trials. Commonly used agents are levamisole, BCG vaccination[33].

Another strategy is passive – specific immunotherapy using monoclonal antibodies especially against CEA antigen and various other tumour antigens which is said to result in improved disease free and overall survival in patient[34]. Of recent interest is MCA 17-1A which resulted in some improvement in Duke stage C patients. Also MCA – attached with tumoricidal drugs used to target potential metastases, local recurrences etc. All are still in experimental stages[35].

TREATMENT OF HEPATIC METASTASES

It is important to biopsy liver metastases for histological diagnosis. Patients with upto 2 or 3 liver mets confined to one lobe of the liver may be offered liver resection. They are not usually resected at the time of colonic surgery. Multiple painful hepatic metastases can be palliated by cytotoxic drugs cryotherapy and laser therapy.

FOLLOW UP OF COLORECTAL CARCINOMA

The objective of follow up is for early detection of recurrent or a metachronous carcinoma that can be treated. The follow up program in general consist of periodic history and physical examination, fecal occult blood (FOB), liver function tests, tumour markers, colonoscopy and radiographic studies. Colonoscopy is an important component of the program. It is a safeguard for detecting anastamotic recurrence, missed synchronous lesions and metachronous tumours at an early stage.

MATERIALS AND METHODS

Cases of colorectal carcinoma were collected from August 2003 to March 2006 for this study. A total of 62 cases were studied of which 14 cases presented as emergencies.

Detailed history was elicited from each patient with special reference to family history, habitations and early symptomatology. Thorough physical examination of the patient was performed for evaluation of general condition, detection of signs and per-rectal examination.

Thorough laboratory investigations were done in all patients, and every patient except those who presented as emergency underwent USG abdomen. Luminal contrast radiographic studies and Colonoscopic evaluation, CT scan abdomen were done in selected cases. Chest skiagram taken for all patients for preoperative evaluation as well as detection of secondaries. Liver function test was done as routine investigation in all patients.

For all possible cases, preoperative biopsy taken via proctoscopic, colonoscopic guidance and histologic type made out before planning treatment. Detailed histopathological reports were available for staging of tumour and assessing the grade of differentiation.

SUMMARY AND RESULTS

In this series of 62 cases of colorectal carcinoma the following observations were made.

Risk factors

Diet

Only one of the 62 patients was pure vegetarian. The majority of patients were illiterate and they could not specify the exact dietary constituents. But most patient gave history of consumption of fat and spicy food. Fibre intake was moderately adequate in most patients.

Tobacco

52 patients out of 62 were using tobacco in some form or other. All the male patients except 4 were regular smokers of beedi or cigarettes. 18 out of 24 female patients were using tobacco in the form of tobacco, pan masala, etc.

INCIDENCE

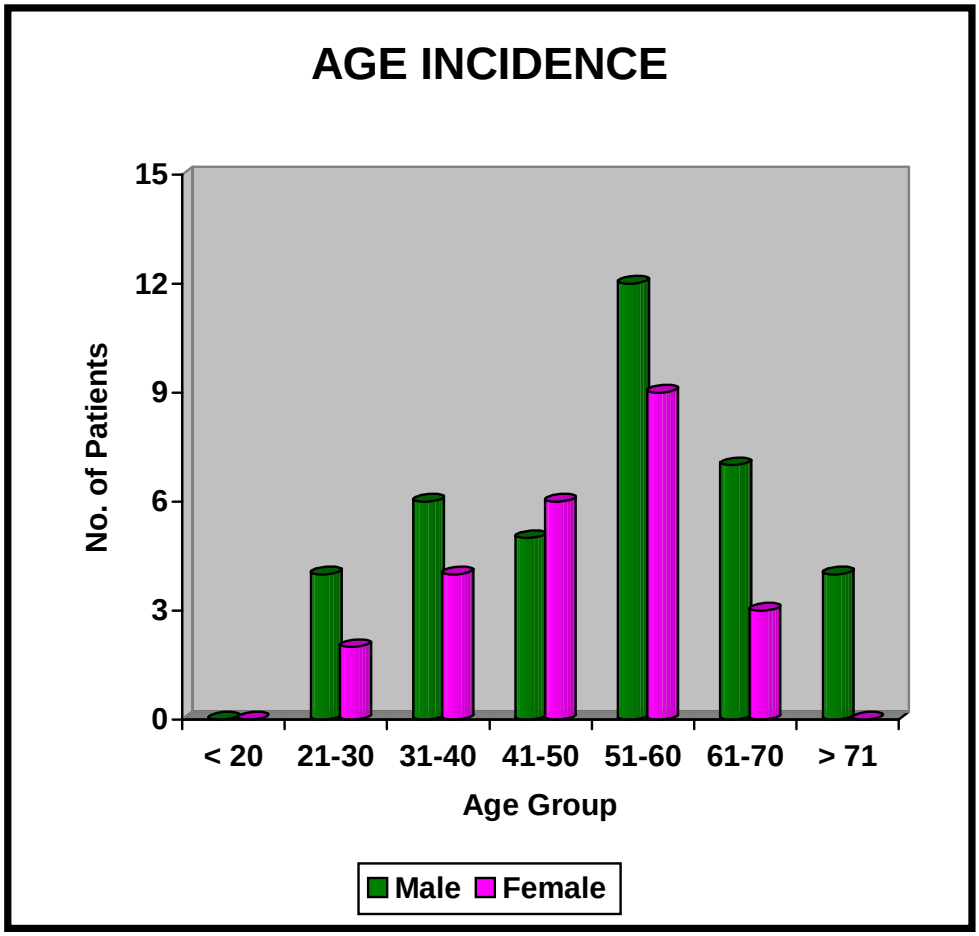
Age

Most of the cases were from older age group. Maximum incidence was in the sixth decade, but even below the age of 30, 6 cases were reported. The tendency to develop colorectal carcinoma is considered to increase progressively with advancing age.

Table No. 1

AGE INCIDENCE

Range (yrs)	Male	Female	Total	Percentage
< 20	0	0	0	0
21-30	4	2	6	9.67
31-40	6	4	10	16.12
41-50	5	6	11	17.74
51-60	12	9	21	33.87
61-70	7	3	10	16.12
> 71	4	0	4	6.45
Total	**38**	**24**	**62**	**100**


AGE INCIDENCE
No. of Patients
15
12
9
6
3
0
< 20
21-30
31-40
41-50
51-60
61-70
> 71
Age Group
Male
Female

Sex

Incidence was relatively more in males. M : F ratio in this series was 1:59:1. The incidence of right colon cancer was nearly equal in both sex but there is definite male preponderance in rectal carcinoma.

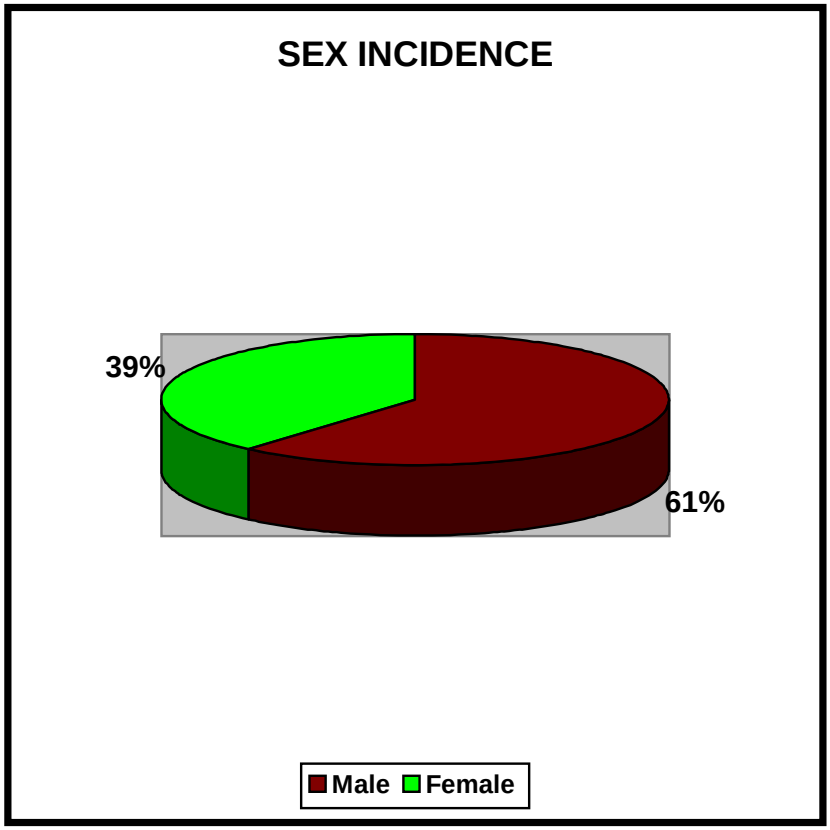
Table No. 2

Sex Incidence

Male	Female
38	24

M : F = 38 : 24

= 1.59 : 1


SEX INCIDENCE
39%
61%
Male
Female

Site

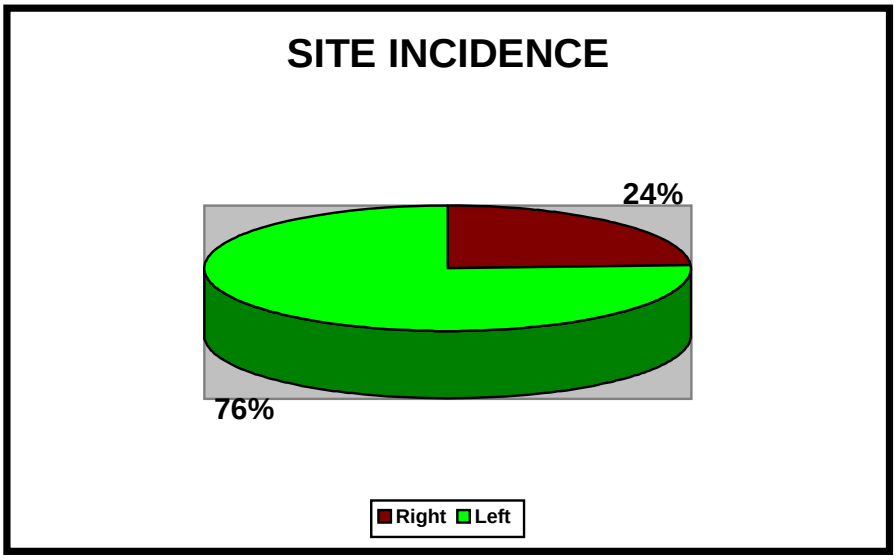
Carcinoma rectum accounted for majority of cases. In 62 patients, 27 were carcinoma of rectum 43.54%. The distribution of tumour in the other sites in the decreasing order of frequency were as follows. Sigmoid colon 9, caecum 6, rectosigmoid junction and transverse colon 4 each, descending colon – 3, ascending colon – 3, and canal – 3, Hepatic flexure – 2, splenic flexure – 1. 15 out of 62 were right sided lesions with 47 being left sided lesion.

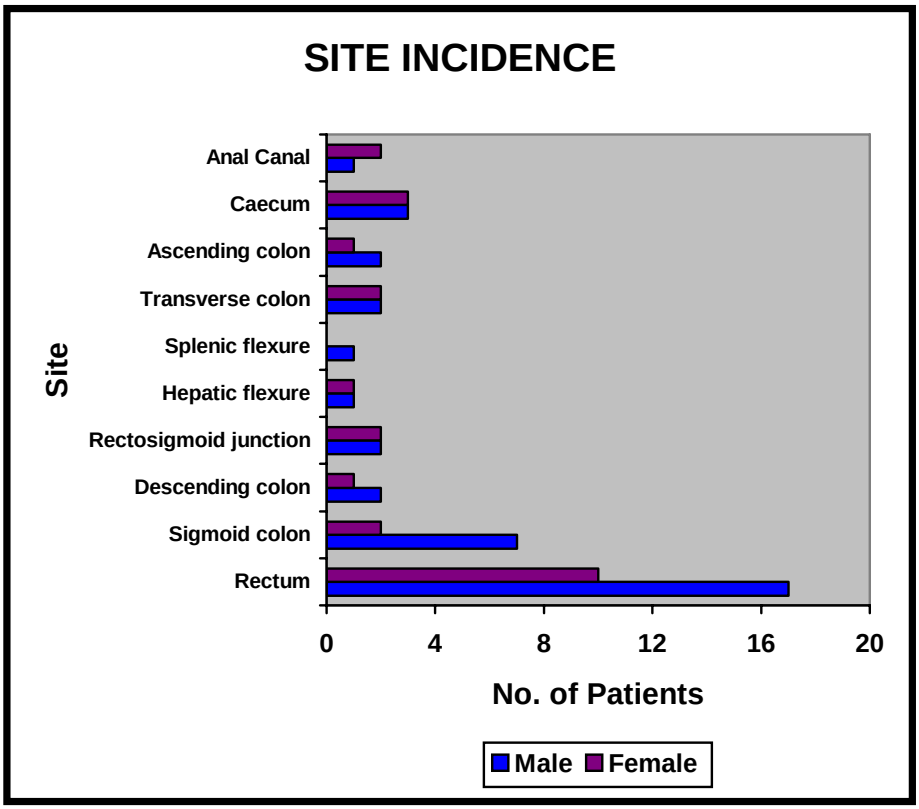
Table No. 3

SITE INCIDENCE

Site	Male	Female	Total	Percentage
Right sided tumour	8	7	15	24.19
Left sided tumour	30	17	47	75.80

Site	Male	Female	Total	Percentage
Rectum	17	10	27	43.54
Sigmoid Colon	7	2	9	14.51
Descending Colon	2	1	3	4.83
Rectosigmoid Junction	2	2	4	6.45
Hepatic Flexure	1	1	2	3.22
Splenic Flexure	1	0	1	1.61
Transverse Colon	2	2	4	6.45
Ascending Colon	2	1	3	4.83
Caecum	3	3	6	9.67
Anal Canal	1	2	3	4.83
Total	**38**	**24**	**62**	**100**


SITE INCIDENCE
24%
76%
Right
Left


SITE INCIDENCE
Anal Canal
Caecum
Ascending colon
Transverse colon
Splenic flexure
Hepatic flexure
Rectosigmoid junction
Descending colon
Sigmoid colon
Rectum
Site
0
4
8
12
16
20
No. of Patients
Male
Female

MODE OF PRESENTATION

Majority of the patients though had symptoms for sometime, tend to ignore them and presented at late stages.

Fourteen of the cases in this series are presented as acute emergencies 22.58%. Twelve of them presented with obstructive features. Out of which 2 were acute on chronic bowel obstruction. In all except 1 were left sided lesion. Two patients presented with features of bowel perforation with peritonitis.

Majority of the cases of right sided colonic tumours had symptoms of altered bowel habits, increasing constipation, bleeding per rectum, being the major complaint. Some patient presented with spurious diarrhea. Majority of bleeding per rectum were seen in rectum and sigmoid lesions. Pain was relatively late symptom. Abdominal lump was present in 2 cases of rectosigmoid growth, 1 case of hepatic flexure growth. Rectal growth was palpated in most cases of carcinoma rectum on per rectal examination.

Transverse colon growth presented with pain abdomen with typical history of diarrhea alternating with constipation. Anemia, anorexia and progressive loss of weight was present in majority of cases at all sites.

Eight patients presented with symptoms of metastasis / disseminated disease. Two presented with skeletal metastases mainly in lumbo dorsal spine out of which one presented with paraplegia. Five cases presented with hepatomegaly, one patient presented with cough, haemoptysis, with multiple lung metastases.

Table No. 4

COLORECTAL CARCINOMA

SIGN AND SYMPTOMS IN THIS SERIES

No. of Cases	R. Colon	L. Colon	Trans. Colon	Sigmoid Colon /Rectosigmoid junction	Rectum / Anal Canal
No. of cases	11	4	4	13	30
Avg. duration of illness	11	6.5	10	7	8
Signs / symptoms					
Pain	7	2	0	3	21
Altered bowel habits	8	4	1	8	18
Bleeding per rectum	2	2	0	8	27
Mucus per rectum	0	2	1	4	14
Tenesmus	1	1	0	5	23
Lump abdomen	4	2	1	2	2
Hepatomegaly		1	0		
Weight Loss	10	3	1	3	10
Loss of appetite	10	3	1	3	10
Lassitude	11	3	0	8	13
Anemia	10	3	1	4	15
Acute obstruction	1		0	3	8
Peritonitis		1	0	0	1

DIAGNOSIS

Diagnosis in this series was not a problem due to late presentation in most of cases. Right sided growth tend to present as mass and left sided growth with features of mass or obstruction and most of rectal growth were palpated except few.

Thorough laboratory investigations were done in all patients, and every patient except those who presented as emergency underwent USG abdomen. Luminal contrast radiographic studies and Colonoscopic evaluation, CT scan abdomen were done in selected cases. Chest skiagram taken for all patients for preoperative evaluation as well as detection of secondaries. Liver function test was done as routine investigation in all patients.

For all possible cases, preoperative biopsy taken via proctoscopic, colonoscopic guidance and histologic type made out before planning treatment. Detailed histopathological reports were available for staging of tumour and assessing the grade of differentiation.

PATHOLOGY AND STAGE

Most of the rectosigmoid and left sided growth were either annular and stenosing or ulcerative with infiltration type. Almost all cases of carcinoma rectum were of ulcerative type. 3 sigmoid growth

showed infiltration into surrounding viscera. 2 cases of carcinoma rectum presented with posterior vaginal wall infiltration. One patient presented with B/L hydronephrosis due to ureteric compression. Majority of right sided growths were polypoidal or cauliflower like growths. One hepatic flexure growth was found to infiltrate into abdomical wall. Two cases of synchronous tumour found, one was synchronous rectal carcinoma and transverse colon growth. While the other was synchronous cystosarcoma phylloides of (R) breast and carcinoma of hepatic flexure. 5 patients presented with distant metastasis. Out of them 3 had multiple liver metastatis, one had dorsolumbar spine metastasis. One patient present with peritoneal metastasis in the form of nodules.

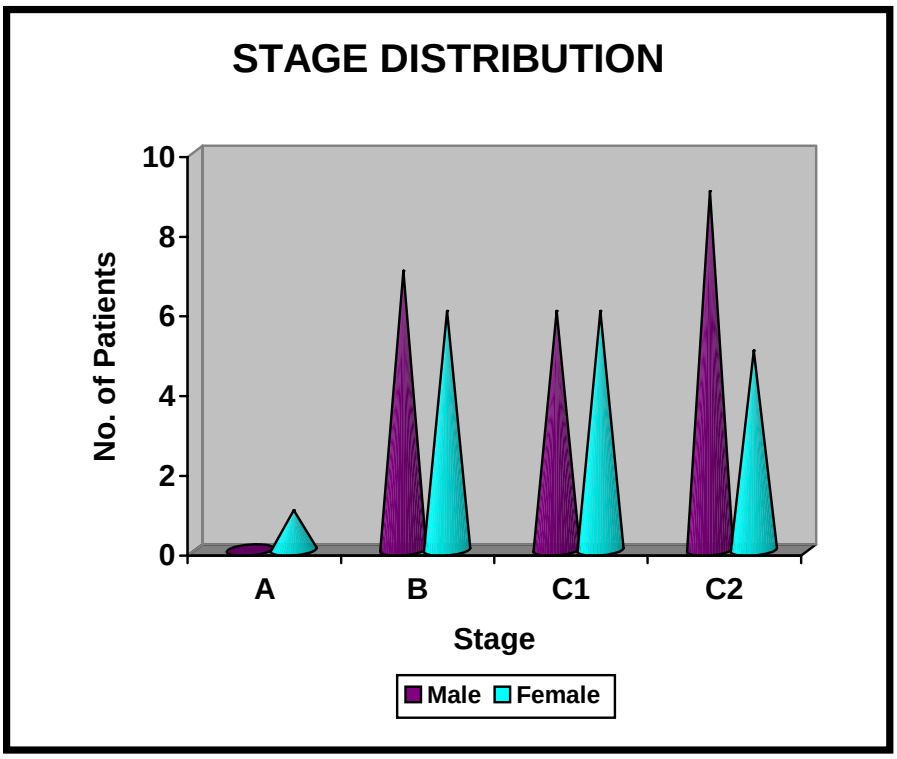
STAGING

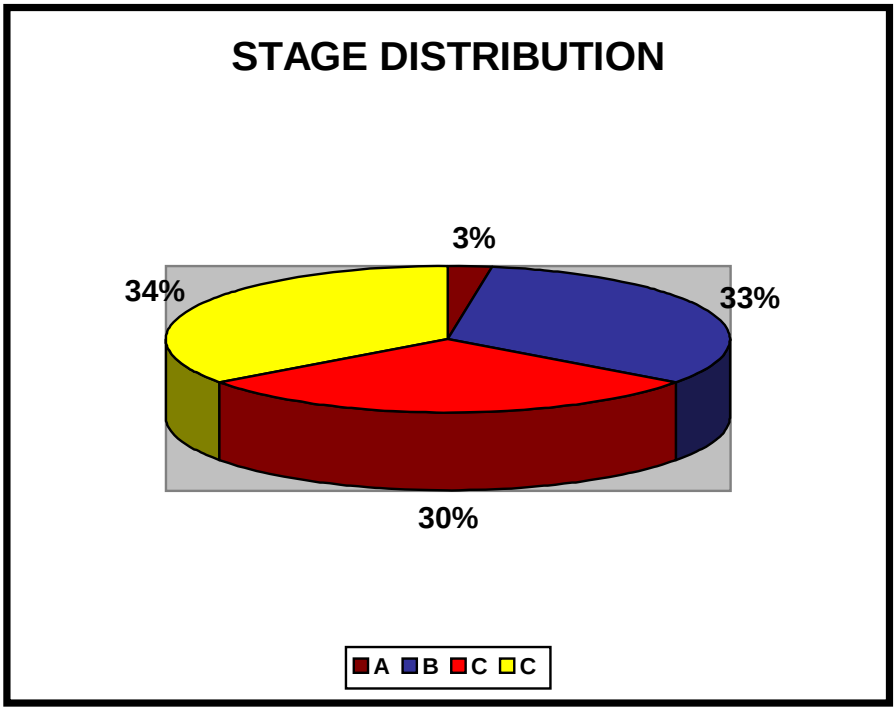
Histopathological staging was carried out on resection specimen and the results were obtained. Only 1 case presented in Duke Stage A (2.5%), 13 were in Stage B (32.5 %, 12 were Stage C_1 (30 %) 14 were stage C_2 (35 %).

Table No. 5

STAGING

Stage	Male	Female	Total	Percentage
A	0	1	1	2.5
B	7	6	13	32.5
C_1	6	6	12	30.0
C_2	9	5	14	35.0
Total	**22**	**18**	**40**	**100**


STAGE DISTRIBUTION
No. of Patients
10
8
6
4
2
0
A
B
C1
C2
Stage
Male
Female


STAGE DISTRIBUTION
3%
34%
33%
30%
A
B
C
C

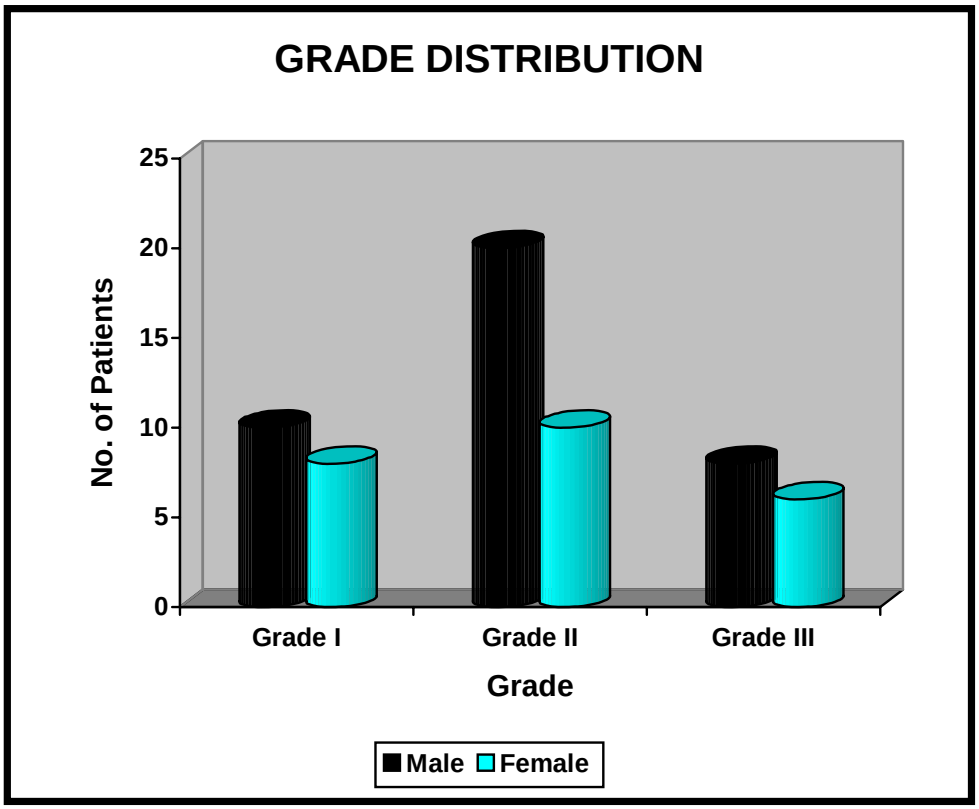
GRADING

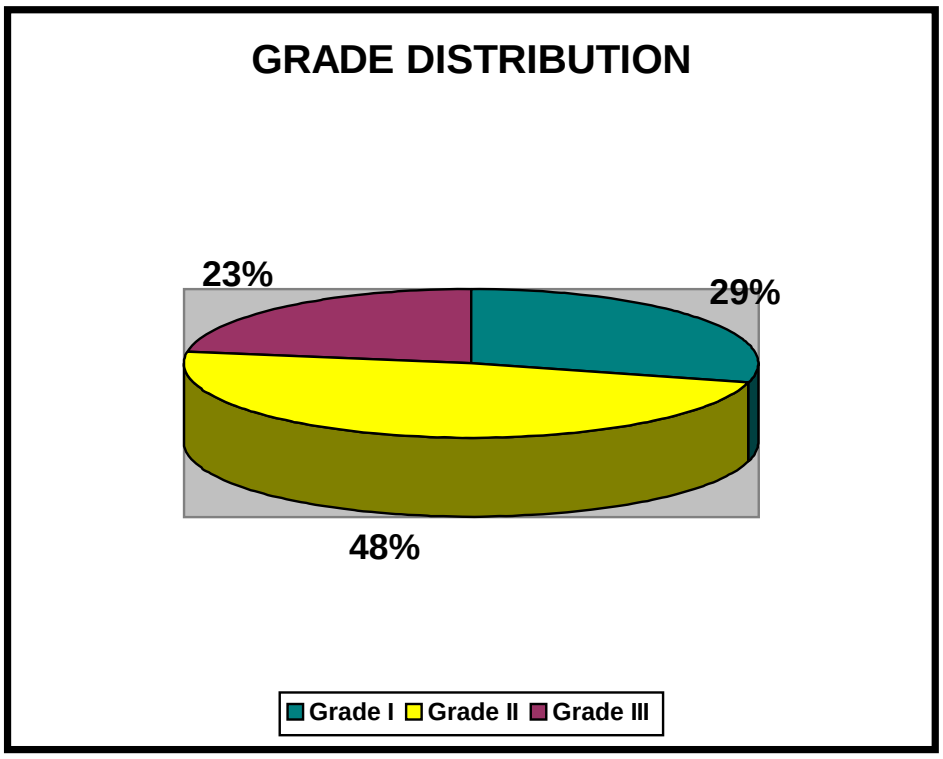
Moderately differentiated tumour predominated. 30 our of 62 cases belonged to this (48.38%) 18 cases were well differentiated (29.03%) and 14 cases were poorly differentiated (22.58%) type.

Table No. 6

GRADING

Grade	Male	Female	Total	Percentage
Grade I	10	8	18	29.03
Grade II	20	10	30	48.38
Grade III	8	6	14	22.58
Total	**38**	**24**	**62**	**100**


GRADE DISTRIBUTION
25
20
15
10
5
0
No. of Patients
Grade I
Grade II
Grade III
Grade
Male
Female


GRADE DISTRIBUTION
23%
29%
48%
Grade I
Grade II
Grade III

SIGMOID LOOP COLOSTOMY

TREATMENT ADOPTED

Surgical resection in the form of either curative or palliative resection or palliative bypass / colostomy was attempted on all patients except, eight. In these eight 3 refused surgery and were treated with CT/RT while remaining went against medical advice.

EMERGENCY CASES

14 patients presenting as emergencies were taken up 2 patients had perforation with peritonitis. One each of descending colon and carcinoma rectum.

The first had presented with perforation near the proximal margin of growth treated with transverse loop colostomy and underwent elective resection and anastomosis in the form of left hemicolectomy. One rectal carcinoma presented with caecal perforation treated with ileostomy and elective APR later.

Twelve cases presenting with bowel obstruction were taken up for emergency surgery. Out of 8 rectal growths 5 were inoperable, treated with Hartman procedure with end colostomy. Remaining were treated with temporary defunctioning colostomy with elective APR later. Out of 3 growths in rectosigmoid / sigmoid colon. 2 were inoperable with extensive local infiltration, treated by transverse loop colostomy. While in one segmental resection (palliative) done with hartman's procedure with elective colorectal anastamosis later. One

caecal growth was inoperable which was treated by ileotransverse anastamosis.

ELECTIVE CASES

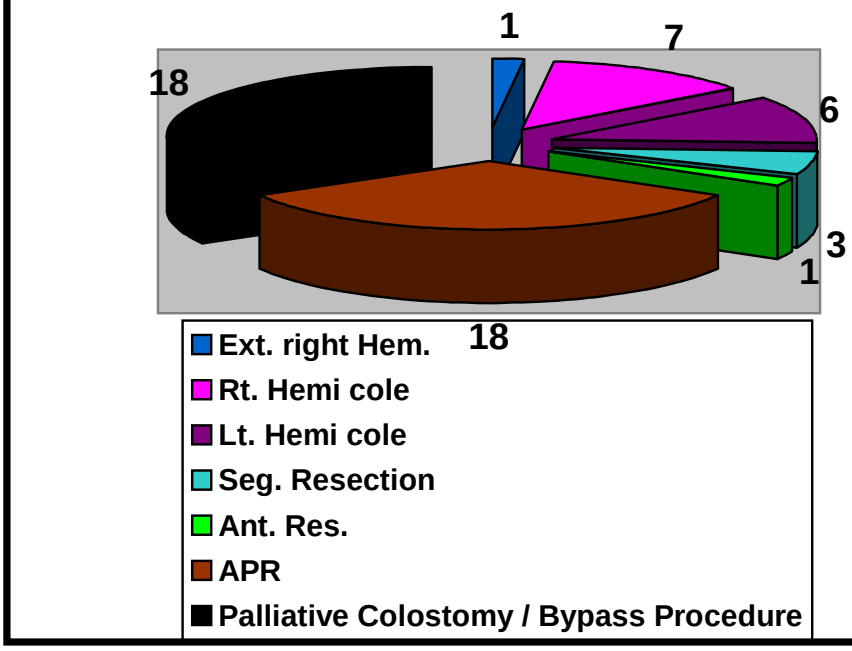
40 patients were treated with elective surgery in whom 12 rectal carcinomas underwent curative APR with one case treated with anterior resection with colo anal anastomosis. 5 cases of left colon growth were treated with left hemicolectomy. 1 patient underwent extended right hemicolectomy for transverse colon growth, and 7 patients were treated with right hemicolectomies for cancer caecum and ascending colon.

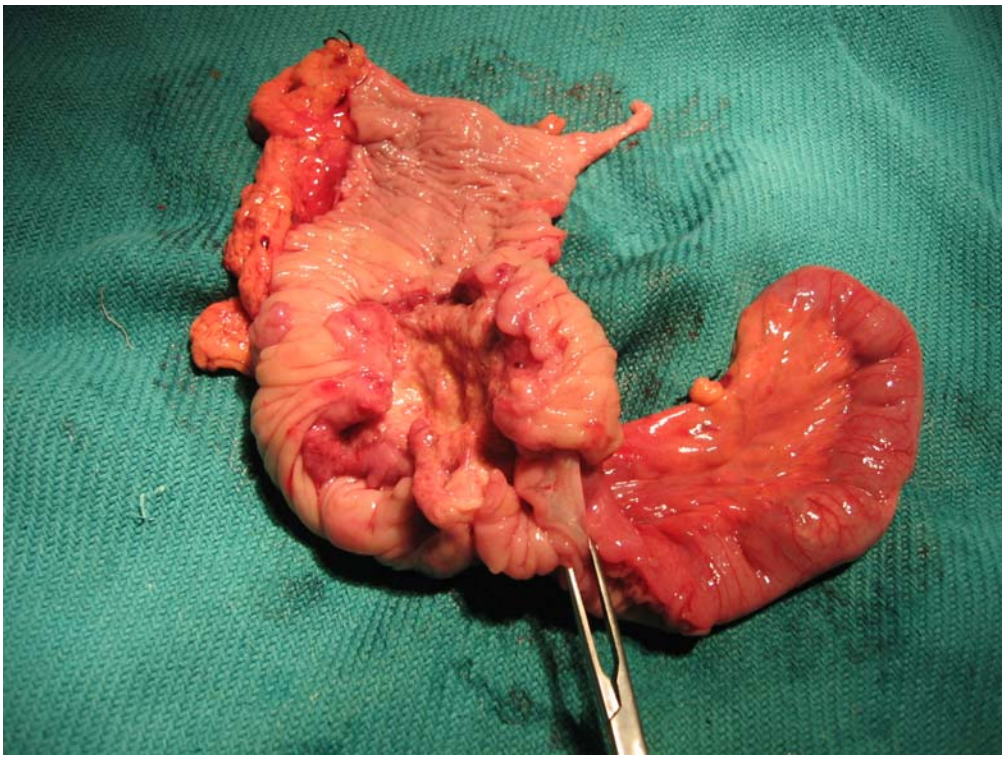
Segmental resection was done in 2 cases, 1 case of sigmoid colon growth and one case of descending colon growth. 2 out of 3 anal canal carcinoma were inoperable, rest treated by APR. Palliative surgery in the form colostomy / by pass procedures were done in 10 cases.

Table No.
SURGICAL PROCEDURE (ELECTIVE & EMERGENCY)

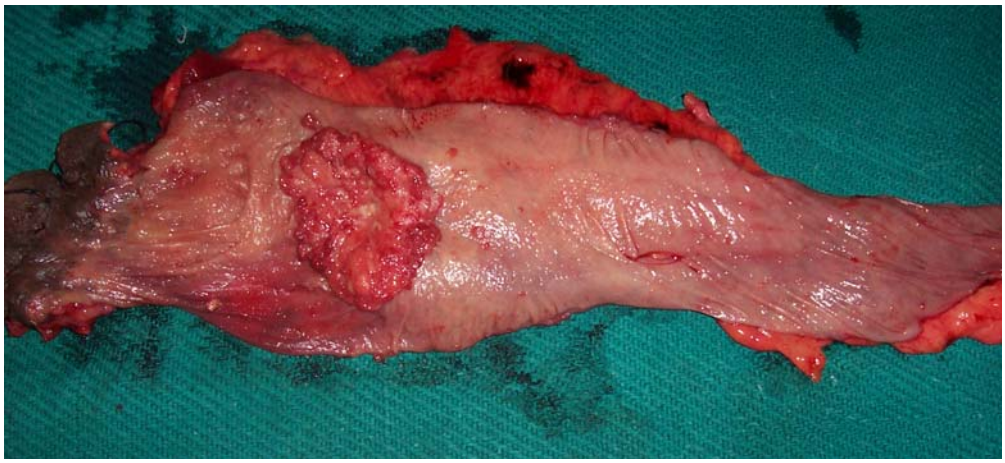
Extended right hemicolectomy	1
Right hemicolectomy	7
Left hemicolectomy	6
Segmental Resection / Anastomosis	3
Anterior Resection	1
APR	18
Palliative Colostomy / Bypass procedure	18

SURGICAL PROCEDURES
(ELECTIVE & EMERGENCY)





RESECTED SPECIMEN – RT HEMI COLECTOMY



RESECTED SPECIMEN – APR

ADJUVANT THERAPHY

Most cases were advised adjuvant chemotheraphy mainly 5 flurourcail and levamisole. A minimum of 6 cycles were given additional 4 cycles were given to partial response patients. 8 people refused further adjuvant therapy. Radiotherapy was given preoperatively to one patient and later underwent Hartman procedure for (carcinoma rectum) obstruction.

COMPLICATIONS

Significant post operative complications like anastamotic leak, faecal fistulae occurred in few patients. Other complications like post operative wound infection occur in few patients.

General complications like post operative respiratory infection, uraemia, electrolyte imbalance were found in some patients. Most of them belonged to older age group with associated diabetes mellitus and poor general status.

FOLLOW UP

Follow up was average with most patients failing to turn up after 3-6 cycles of chemotherapy, 8 patients developed multiple hepatic metastases during follow up. While 3 patients had local recurrence. 18 patients have regular follow up with repeat USG and basic blood investigations being normal.

CONCLUSION

- Incidence of colorectal carcinoma is on the increase in the past few years.
- Most patients were non vegetarians and taking fat diet. But no definite evidence of diet as a risk factor could be identified.
- Commonest age group of colorectal cancer was on the 6^{th} decade.
- Incidence was higher in males, M : F was 1.59 : 1
- Incidence of tobacco abuse was high in most of the patients
- Rectum was the most commonest site of large bowel (carcinoma 43.54%) in the series.
- Most of the patients tend to present late even though they have been symptomatic for sometime. This is evidenced by the fact that only 35% of the patients belonged to Duke Stage A & B. Distant metastases were present in 5 patients at time of diagnosis.
- 22.58% presented with acute emergency in the form of bowel obstruction or perforation.
- Surgery is the main stay of treatment whether palliative or curative.

- To conclude it may be said that over the age of 40 years when a patient complains of altered bowel habits or bleeding per rectum every efforts should be made to thoroughly investigate him to find out if he harbours malignancy of large bowel. The prognosis is poor in cases presenting late.
- The take home point is early recognition, adequate assessment and early surgery have gratifying results to the surgeon and patient alike.

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PROFORMA FOR STUDY OF COLORECTAL MALIGNANCIES

Name : **I.P. No.** **Case No.**

Age & Sex : **Unit :**

I. PRESENTING COMPLAINTS

1	Pain Abdomen		
2	Bleeding per rectum		
3	Tenesmus		
4	Abdominal Distension		
5	Alteration in Bowel Habits		
6	Mucus Per rectum		
7	Lump abdomen		
8	Lassitude		
9	Loss of appetite		
10	Loss of weight		
11	Others		

II. PRESENT HISTORY :

III. PAST HISTORY :

IV. PERSONAL HISTORY :

1	High fat diet		
2	Vegetarian / Non Vegetarian		
3	Smoker / tobacco consumer		
4	Alcoholic		
5	Betel nut Chewer		

V. TREATMENT HISTORY :

VI. GENERAL EXAMINATION

1	Built		
2	Nourishment		
3	Performance Status		
4	Pallor		
5	Ictarus		
6	Clubbing		
7	Cyanosis		
8	Pedal Edema		
9	Lymphadenopathy		

VII. VITAL SIGNS

PR. :

BP. :

VIII. LOCAL EXAMINATION :

P/R EXAM :

P/V EXAM :

IX. OTHER SYSTEMS :

CVS -

RS –

X. SPECIALIST OPINION :

XI. BASIC INVESTIGATIONS :

COMPLETE HEMOGRAM :

HB - %

TC - / Cu. Mm

DC - P L E B M

ESR - mm / Hr

PC - / Cu. Mm

BLOOD : **URINE :**

UREA - **mg %** **ALB -**

SUGAR - **mg %** **SUGAR -**

DEPOSITS

SERUM : **MOTION :**

CREATININE - **mg %** **OCCULT BLOOD -**

LFT :

BILIRUBIN (T) - mg % **PROTEINS (T) - gm %**

DIRECT - mg % **ALBUMIN - gm %**

INDIRECT - lu/Lit **GLOBULINE - gm %**

AST (SGOT) - lu/Lit

ALT (SGPT) - lu/Lit

SAP - lu/Lit

ECG -

CXR PA VIEW -

X RAY ABDOMEN –

XII. SPECIAL INVESTIGATIONS :

BARIUM SERIES :

USG ABDOMEN :

COLONSCOPY :

CT SCAN ABDOMEN :

BIOPSY REPORT :

XIII. DIAGNOSIS :

XIV. TREATMENT :

XV. SPECIMEN HPE REPORT :

XVI. FOLLOW UP :

MASTER CHART

Sl. No.	Name	Age / Sex	IP No.	Risk Factors		Mode of Presentation	Investigations				Site of tumour	Surgery	HPE
				Smoking/ tobacco	Fat intake		Blood	Barium Study	Colono scopy	CT Scan			
1	Magesh	22 / M	62141	+	M	NE	+	+	+	+	RSJ	PS	AC
2	Kaleeswari	36 / F	73189	+	M	NE	+	-	+	+	R	APR	MAC
3	Vairaperumal	74 / M	75108	+	H	E	+	-	-	-	R	APR	AC
4	Thangavel	63 / M	79006	+	M	NE	+	+	+	+	TC	PS	AC
5	Indirani	48 / F	80014	-	H	NE	+	-	+	+	AC	RH	MAC
6	Velingiri	75 / M	86203	+	M	NE	+	-	+	+	R	APR	AC
7	Rayathal	58 / F	1012	+	M	NE	+	+	-	+	AC	RH	AC
8	Velliammal	53 / F	2399	+	M	E	+	-	-	-	RSJ	PS	AC
9	Karuppathal	64 / F	7506	+	M	NE	+	-	+	+	S	CT	AC
10	Dhanalakshmi	49 / F	10119	+	L	E	+	-	-	-	R	PS	MAC
11	Joseph	26 / M	12215	-	H	NE	+	-	+	+	R	AR	AC
12	Sarojini	56 / F	17308	+	L	NE	+	+	+	+	RSJ	PS	MAC
13	Arayee	64 / F	18012	+	M	NE	+	+	-	+	R	APR	MAC
14	Santhabeebi	57 / F	20117	+	H	NE	+	+	+	+	TC	PS	MAC
15	Noorjahan	26 / F	21206	-	H	NE	+	+	+	+	C	RH	AC
16	Munusamy	59 / M	26508	+	M	E	+	-	+	+	R	APR	AC
17	Veluraj	44 / M	28875	+	M	NE	+	-	-	-	R	APR	AC
18	Kuppuasmy	69 / M	30147	+	H	NE	+				A	APR	MAC
19	Balathandapani	32 / M	30680	-	M	E	+	-	-	-	R	PS	AC

Sl. No.	Name	Age / Sex	IP No.	Risk Factors		Mode of Presentation	Investigations				Site of tumour	Surgery	HPE
				Smoking/ tobacco	Fat intake		Blood	Barium Study	Colono scopy	CT Scan			
20	Kuppusamy	53 / M	31508	+	M	NE	+	+	+	+	SF	PS	AC
21	Marappan	55 / M	34215	+	H	NE	+	-	+	+	R	APR	AC
22	Chellammal	39 / F	34508	+	M	NE	+	-	+	-	HF	AMA	AC
23	Manivel	52 / M	37114	+	L	E	+	-	-	-	C	PS	MAC
24	Sarojo	55 / F	38006	-	M	NE	+	+	+	+	TC	PS	AC
25	Dhanraj	38 / M	43508	-	M	NE	+	-	+	-	S	AMA	AC
26	Veerappan	51 / M	44001	+	H	NE	+	+	+	+	R	PS	AC
27	Ponnusamy	58 / M	45898	+	L	E	+	-	-	-	S	PS	MAC
28	Marappan	78 / M	49909	+	M	NE	+	+	-	+	R	APR	MAC
29	Nagamani	48 / F	52605	+	M	NE	+	-	-	+	A	RT	SC
30	Moorthi	35 / M	56817	+	M	NE	+	+	+	+	DC	LH	AC
31	Sathya Bama	35 / F	59007	+	H	E	+	-	-	-	R	PS	AC
32	Sivakami	44 / F	53071	-	L	NE	+	+	+	+	R	APR	AC
33	Sekar	25 / M	70080	+	M	NE	+	-	+	+	S	LH	MAC
34	Natarajan	43 / M	72399	+	M	NE	+	+	+	+	R	PS	AC
35	Muthusamy	57 / M	75935	+	H	NE	+	+	+	+	C	RH	MAC
36	Ganesan	37 / M	76005	+	M	NE	+	-	+	+	S	LH	AC
37	Thangathal	54 / F	78006	+	M	E	+	-	-	-	S	LH	AC
38	Ramayal	58 / F	2024	+	M	NE	+	+	+	+	R	APR	AC
39	Selvaraj	61 / M	4007	+	L	NE	+	+	+	+	S	LH	AC
40	Maragatham	41 / F	6985	+	H	E	+	-	-	-	R	APR	AC

Sl. No.	Name	Age / Sex	IP No.	Risk Factors		Mode of Presentation	Investigations				Site of tumour	Surgery	HPE
				Smoking/ tobacco	Fat intake		Blood	Barium Study	Colono scopy	CT Scan			
41	Paneerselvam	49 / M	10250	+	H	NE	+	+	+	+	C	RH	MAC
42	Kannan	35 / M	10972	+	M	NE	+	-	-	-	R	PS	AC
43	Mariammal	49 / F	12506	+	M	NE	+	-	-	+	A	PS	M
44	Sivasankar	34 / M	14980	+	M	NE	+	+	+	+	R	APR	AC
45	Renuka Devi	29 / F	17008	-	M	NE	+	+	-	-	R	AMA	MAC
46	Sevalappan	68 / M	20015	+	L	E	+	-	-	-	R	PS	AC
47	Chellammal	63 / F	22580	+	H	NE	+	+	+	+	C	RH	AC
48	Meenatchi	59 / F	24917	-	M	NE	+	+	+	+	R	CT	MAC
49	Mani	24 / M	27090	+	H	NE	+	-	+	+	S	LH	MAC
50	Gnanambal	57 / F	3001	+	L	E	+	-	-	-	DC	SR	MAC
51	Periyasamy	58 / M	33596	+	M	NE	+	-	-	+	R	APR	AC
52	Dhurairaj	55 / M	36040	+	M	NE	+	+	+	+	TC	EHC	AC
53	Angappan	73 / M	37984	-	M	NE	+	+	-	+	S	SR	AC
54	Palaniammal	37 / F	39585	+	M	NE	+	-	+	+	R	APR	AC
55	Palanivel	59 / M	44780	+	M	E	+	-	-	-	R	APR	AC
56	Devanbu	46 / M	47963	+	H	NE	+	+	+	+	AC	PS	MAC
57	Rangasamy	53 / M	51647	+	M	NE	+	+	+	+	DC	SR	MAC
58	Kamaraj	66 / M	54964	+	M	NE	+	+	+	+	R	APR	AC
59	Vadivel	52 / M	59072	+	H	NE	+	+	+	+	C	RH	AC
60	Ganesan	47 / M	61289	+	M	NE	+	+	+	-	HF	AMA	AC
61	Kalisamy	63 / M	64712	+	M	NE	+	+	+	+	R	APR	AC

Sl. No.	Name	Age / Sex	IP No.	Risk Factors		Mode of Presentation	Investigations				Site of tumour	Surgery	HPE
				Smoking/ tobacco	Fat intake		Blood	Barium Study	Colono scopy	CT Scan			
62	Selvaraj	65 / M	65584	+	M	NE	+	-	+	-	RSJ	AMA	MAC

E - Emergency

NE - Non Emergency

Site

R - Rectum

S.C. - Sigmoid Colon

D.C. - Descending Colon

R.S.J. - Recto Sigmoid Junction

H.F. - Hepatic Flexure

S.F. - Splenic Flexure

T.C. - Transverse Colon

A.C. - Ascending Colon

C - Caecum

Ac - Anal Canal

Surgery

APR - Abdominoperineal Resection

AR - Anterior Resection

SR - Segmental Resection

RH - Right Hemicolectomy

LH - Left Hemicoletomy

ERH - Extended Right Hemicolectomy

P.S. - Palliative Surgery

HPE

AC - Adenocarcinoma

M - Melanoma

SC - Squamous cell carcinoma

MAC - Mucoid adenocarcinoma

Fat Intake

H - High

M - Moderate

L - Low

Treatment

CT - Chemotherapy

RT - Radiotherapy

AMA - Against Medical Advice